

DEC 2020: Table of Contents

Paper 1	5
1. a) Fetal circulation with diagrammatic illustration and changes after birth.....	5
1. b) Ductus-dependent congenital heart diseases.....	9
2. Discuss the role of the following in health and diseases a) Copper. b) Zinc. c) Magnesium.....	12
3. a) Describe the evolutionary development of the hemoglobin's from embryo to adult. 15	
3. b) Describe the relationship among the various hemoglobin's in first year of life and their implication in diagnosing hereditary hemoglobinopathies.....	18
4. a) Illustrate the pathophysiology of R-L shunt.	19
5. a) Development of diaphragm, trachea and esophagus and types of TOF with diagrams.	21
5. b) Briefly describe the important attributes of various patterns of genetic inheritance .	24
6. List the various study designs for biomedical research with one suitable example for each	28
7. a) Briefly describe the indicators of child health	29
7. b. List the major programmatic interventions to reduce the IMR.....	32
8.a) a) Chi Square test (illustrate with an example a setting where it should be used)	36
8.b Quality Improvement Initiatives in healthcare (illustrate with an example).	40
9. Discuss salient features of management of children with tuberculosis as per National Tuberculosis Elimination Programme (NTEP).....	43
10. a) What is child labour and discuss the measures to control it in the society?	45
10. b) How will you handle the Cyber bullying in children?	47
Paper 2	50
1. a) Algorithm of neonatal resuscitation programme (NRP).....	50
1. b) Steroid resistant nephrotic syndrome.	51

2. a) Bone age assessment and physical growth in children.	53
2. b) Point of Care (POC) tests for the diagnosis of Urinary Tract Infections (UTIs).....	59
3.a) Management of transfusion-dependent thalassemia (TDT)	61
3. b) Diagnosis of tuberculous meningitis.....	65
4. a) Overview of learning disability disorders.	69
4. b) Craniosynostosis	70
5. a) Approach to a child with recurrent wheezing.	73
5. b) Treatment of nutritional rickets.....	76
6. a) Staging & management hypoxic ischemic encephalopathy (HIE).....	78
6.b) Approach to a newborn with respiratory distress	81
7. a) Management of neonatal seizures.	85
8. a) Principles of management of autistic spectrum disorders	91
9. a) Clinical presentation & management of Henoch-Schonlein purpura.....	94
9. b) Risk stratification in Acute Lymphoblastic Leukemia (ALL) in children.....	96
10. a) Autoimmune encephalitis.....	98
10. b) Immunization in adolescents	102
Paper 3	106
1. a) Approach to child with upper airway obstruction	106
1. b) Types of supraventricular tachycardia and management of SVT with circulatory failure	109
2.a) Evaluation of medical morbidities in Down syndrome.	112
2. b) Management of septic shock.....	114
3.a) Diagnosis of Wilson's disease.....	117
3. b) Emergency triage and treatment in pediatric practice	119
4. a) Importance of fundus examination in children in clinical practice.	121
4. b) Psychosocial screening in adolescents.....	123

5. a) Differential diagnosis of fever with rash in children	125
5. b) Discuss the endocrine causes and its evaluation of short stature.	128
6. a) Complications of severe falciparum malaria.....	130
6. b) Prevention of overweight and obesity in adolescents.	134
7. a) Indications of closure of ventricular septal defects in children.....	136
8. a) Management of scorpion sting.	140
8. b) Organ donation.....	142
9. a) Role of CBC in pediatric OPD.....	146
9. b) Drug surveillance.....	149
10. a) Management of a child with Kawasaki disease	152
10. b) Supratentorial intracranial tumors in children.....	154
Paper 4.....	157
1. a) Management of acute lung injury.....	157
1. b) Metabolic autopsy.....	160
2. a) Prenatal diagnosis of genetic disorders.	163
2. b) Hyperoxia test	166
3. a) Oxygen dissociation curve and its clinical implication	169
3. b) Importance of Human milk banking.....	171
4. a) Intergenerational cycle of malnutrition	173
4. b) Rapid sequential intubation.	176
5. a) Immunomodulators in pediatric patients.....	178
5. b) Cyberbullying.....	181
6. a) Modes of ventilation in pediatric patients.....	182
6. b) Point of care ultrasonography in PICU	185
7. a) Modified Glasgow Coma Scale.....	188
7. b) Pediatric multisystem inflammatory syndrome.....	190

8. a) Pediatric pain management.....	194
8. b) Counseling in chronic pediatric diseases	197
9. a) Plasmapheresis in pediatric practice.	199
9. b) Principles of gene therapy	202
10. a) Preventing COVID-19 infection	205
10. b) Indications of IVIG in pediatric practice	207



Paper 1

1. a) Fetal circulation with diagrammatic illustration and changes after birth.

- ✚ Fetal circulation is a unique system that allows a fetus to receive oxygen and nutrients from the mother's blood supply while bypassing certain organs that aren't fully functional before birth.
- ✚ After birth, significant changes occur as the baby starts breathing and the organs take over functions previously managed by the mother's body.
- ✚ The transition from fetal to postnatal circulation is a critical process that ensures proper oxygenation and systemic perfusion in the newborn
- ✚ Any disruptions in this transition can lead to cardiovascular complications in neonates.

Fetal Circulation

1. Placental Gas Exchange: In utero, oxygen and nutrients are supplied to the fetus via the placenta. The fetal lungs are non-functional, and the liver plays a minor role in metabolic functions.

2. Umbilical Cord: The umbilical cord contains two arteries and one vein. Oxygenated blood and nutrients are transported from the placenta to the fetus via the umbilical vein, while deoxygenated blood and waste products are carried away by the umbilical arteries.

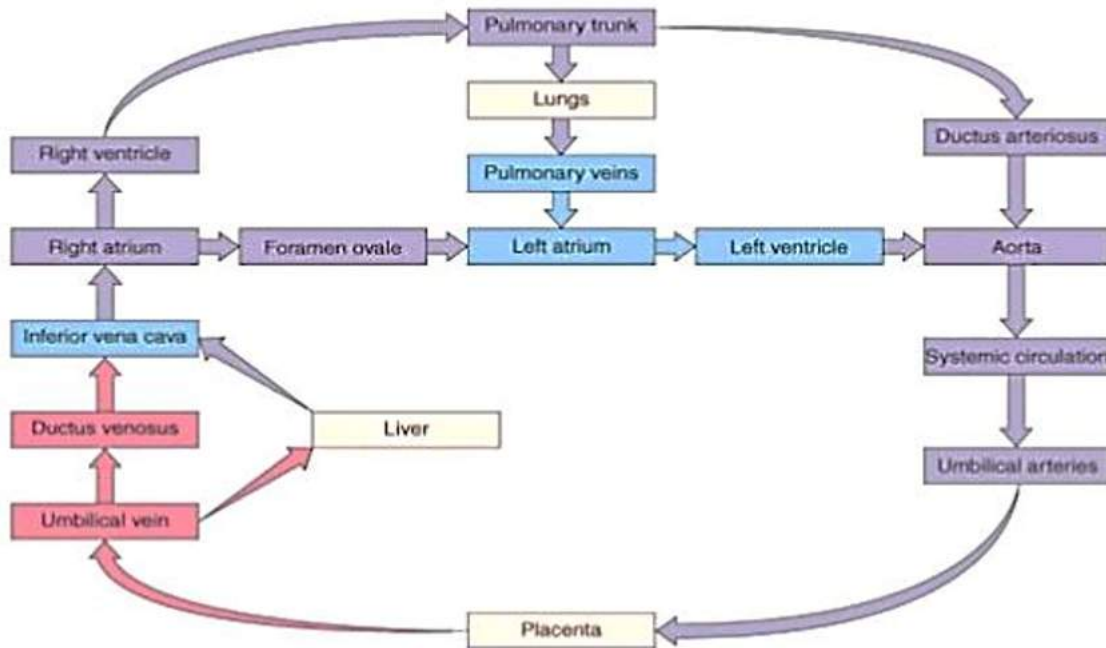
3. Ductus Venosus: In the fetal liver, blood flows through the ductus venosus, which shunts a portion of oxygenated blood directly into the inferior vena cava, bypassing the liver.

4. Foramen Ovale: Blood entering the right atrium is directed through the foramen ovale, a hole in the atrial septum, into the left atrium. This bypasses the non-functional fetal lungs.

5. Ductus Arteriosus: Blood from the right ventricle is pumped into the pulmonary artery but is shunted away from the lungs through the ductus arteriosus and into the aorta. This further ensures that most of the blood bypasses the lungs.

6. Umbilical Blood Oxygenation: Oxygenated blood from the placenta mixes with deoxygenated blood in the right atrium but is mostly directed towards the foramen ovale, preserving a higher oxygen content.

8. Umbilical Arteries: Return deoxygenated blood and waste products from the fetus to the placenta.



1. Lung Expansion: The first breath taken by the newborn triggers lung expansion. This leads to an increase in pulmonary vascular resistance and a decrease in systemic vascular resistance due to increased oxygen in the lungs.

3. Closure of Ductus Arteriosus: Increased oxygen levels cause the muscular wall of the ductus arteriosus to contract, gradually closing off this vessel. Closure is typically complete within the first few days of life.

4. Ductus Venosus Closure: With the interruption of umbilical blood flow, the ductus venosus constricts and eventually closes.

5. Liver Function: As the newborn begins to rely on the gastrointestinal tract for nutrient absorption, liver function increases, and the liver gradually takes on its metabolic role.

6. Systemic Blood Flow: The systemic circulation adapts to the increased oxygen content, and the neonate's systemic blood flow becomes independent of umbilical circulation.

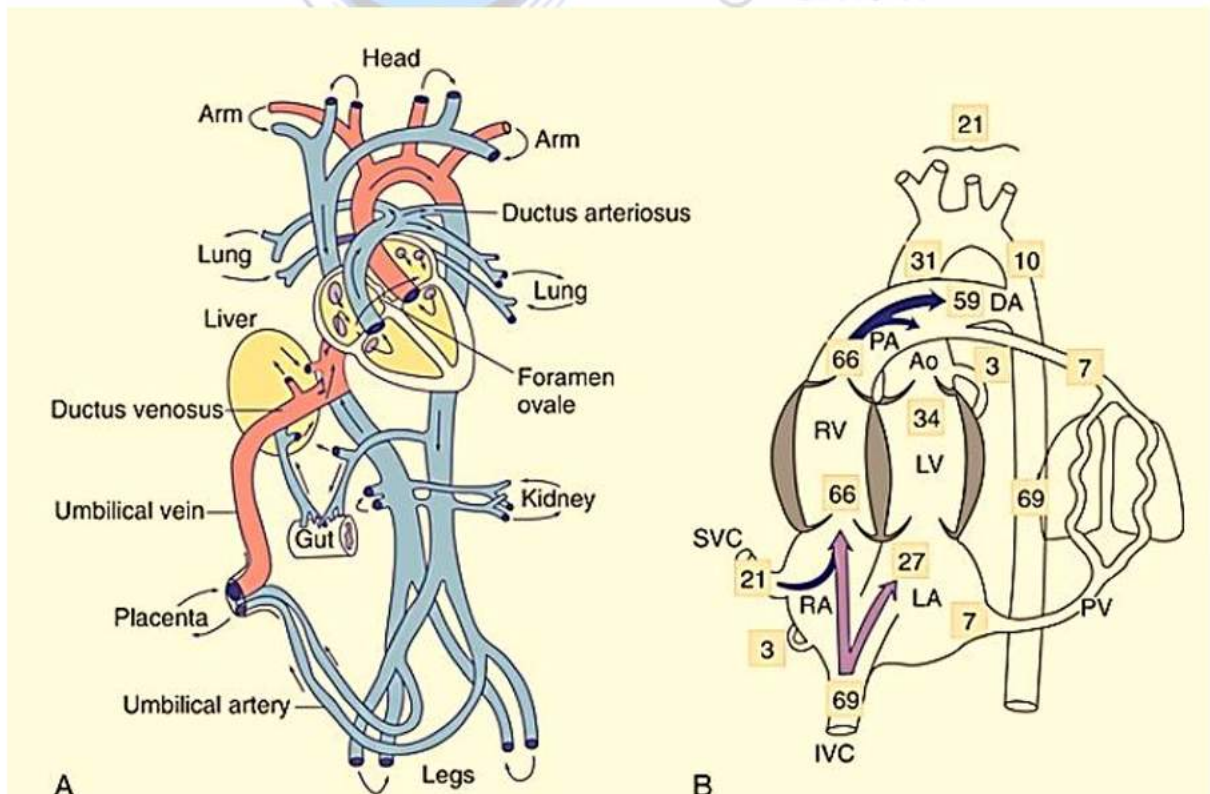
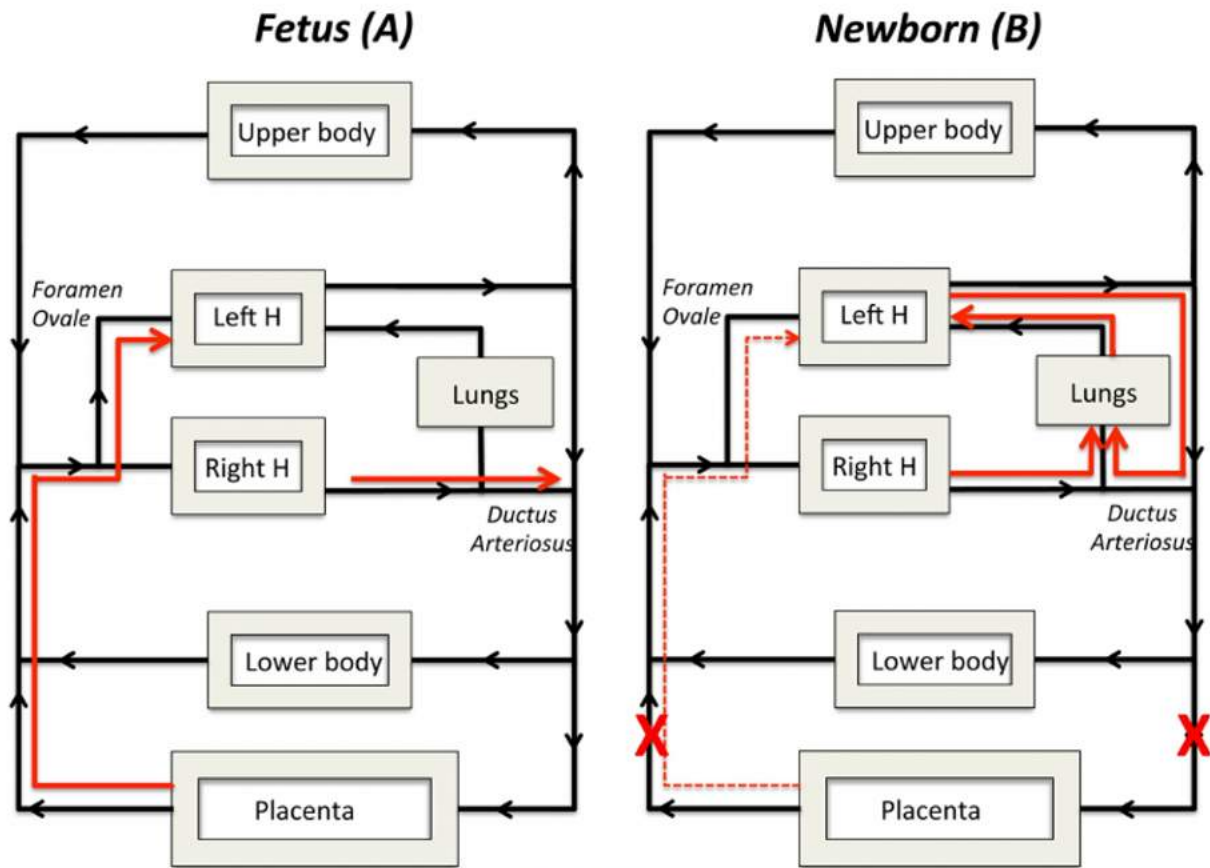
7. Maintenance of Umbilical Blood Flow: In some cases, the umbilical arteries and ductus venosus may remain patent for a short period, but they gradually close within a few days to weeks after birth.

8. Changes in Circulation: The heart and circulatory system start functioning independently. The right side of the heart pumps deoxygenated blood to the lungs, and the left side pumps oxygenated blood to the rest of the body.

9. Degeneration of Umbilical Vessels: The umbilical vein and arteries close and eventually become ligamentous structures (round ligament of the liver and umbilical ligaments).

10. Continued Maturation: Over the first few weeks to months, the cardiovascular system continues to mature, and circulation becomes similar to that of an adult.





A- Diagrammatic representation of Fetal circulation; B- Oxygen tensions (mm Hg)

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1. b) Ductus-dependent congenital heart diseases.

Ductus-Dependent Congenital Heart Diseases:

Pathophysiology:

1. **Ductus Arteriosus in Fetal Circulation:** In fetal life, the ductus arteriosus is a vital conduit that diverts blood from the pulmonary artery to the aorta, bypassing the non-functioning fetal lungs.
2. **Transition at Birth:** Normally, it closes shortly after birth as the newborn's lungs take over the oxygenation of blood.
3. **Dependence in Certain Conditions:** In specific congenital heart diseases, the closure of the ductus arteriosus can be life-threatening due to the reliance on it for either systemic or pulmonary circulation.

Ductal-Dependent Systemic Blood Flow Conditions:

1. **Hypoplastic Left Heart Syndrome (HLHS):**
 - **Pathophysiology:** Underdevelopment of the left heart structures, leading to inadequate systemic circulation.
 - **Clinical Features:** Cyanosis, lethargy, and rapid breathing. Heart failure symptoms may develop as the ductus closes.
 - **Management:** Prostaglandin E1 to maintain ductal patency, followed by staged surgical procedures (Norwood, Glenn, and Fontan operations).
2. **Critical Aortic Stenosis and Coarctation of the Aorta:**
 - **Pathophysiology:** Obstruction to blood flow from the left ventricle.
 - **Clinical Features:** Heart failure, poor feeding, and shock.
 - **Management:** Balloon valvuloplasty for aortic stenosis, surgery for coarctation.

Ductal-Dependent Pulmonary Blood Flow Conditions:

1. **Tetralogy of Fallot with Pulmonary Atresia:**
 - **Pathophysiology:** Combination of defects leading to severely reduced pulmonary blood flow.

- **Clinical Features:** Severe cyanosis, clubbing of fingers, and spells of deep cyanosis (Tet spells).
- **Management:** Prostaglandin E1, shunt surgery, and complete repair in infancy.

2. **Tricuspid Atresia and Ebstein Anomaly:**

- **Pathophysiology:** Abnormalities leading to reduced blood flow to the lungs.
- **Clinical Features:** Cyanosis, dyspnea, and fatigue.
- **Management:** Prostaglandin E1, followed by surgical interventions like shunt placement or Fontan procedure.

Diagnostic Modalities:

1. **Echocardiography:** Gold standard for diagnosis, providing detailed information about the anatomy and function of the heart.
2. **Cardiac Catheterization:** Used for both diagnostic and interventional purposes.
3. **Pulse Oximetry and Blood Gases:** To assess the extent of hypoxemia.

Advanced Management Strategies:

1. **Pharmacological Management:** Besides prostaglandin E1, diuretics, inotropes, and afterload reducers may be used.
2. **Surgical Interventions:** Depending on the condition, various surgical techniques are employed, ranging from palliative shunts to complex reconstructive surgeries.
3. **Long-term Care:** Regular follow-up with a pediatric cardiologist, attention to growth and development, and management of complications like arrhythmias or heart failure.

Long-term Implications:

1. **Neurodevelopmental Outcomes:** Children with complex congenital heart disease are at risk for developmental delays and require ongoing surveillance and support.
2. **Lifelong Follow-up:** Many of these conditions require lifelong cardiac care, including potential reoperations or interventions in adulthood.

3. **Quality of Life:** With advances in surgical and medical care, many children with these conditions can lead active lives, though some may have limitations depending on the severity of their condition.

Duct-Dependent Condition	Pathophysiology	Clinical Presentation	Management
Ductal-Dependent Systemic Blood Flow			
<i>Hypoplastic Left Heart Syndrome (HLHS)</i>	Underdevelopment of left heart structures, inadequate systemic circulation	Cyanosis, lethargy, rapid breathing, heart failure symptoms as ductus closes	Prostaglandin E1, staged surgical procedures (Norwood, Glenn, Fontan)
<i>Critical Aortic Stenosis</i>	Severe narrowing of the aortic valve, obstructing left ventricular outflow	Heart failure, poor feeding, shock	Prostaglandin E1, balloon valvuloplasty, possible surgery
<i>Coarctation of the Aorta</i>	Narrowing of the aorta, obstructing blood flow	Heart failure, poor feeding, shock, differential blood pressure in limbs	Prostaglandin E1, surgical repair
<i>Interrupted Aortic Arch</i>	Discontinuity of the aortic arch	Heart failure, poor feeding, shock	Prostaglandin E1, surgical repair
Ductal-Dependent Pulmonary Blood Flow			
<i>Tetralogy of Fallot with Pulmonary Atresia</i>	Combination of defects causing reduced pulmonary blood flow	Severe cyanosis, clubbing, Tet spells	Prostaglandin E1, shunt surgery, complete repair
<i>Critical Pulmonary Stenosis/Atresia</i>	Severe narrowing or closure of the pulmonary valve	Cyanosis, heart failure, tachypnea	Prostaglandin E1, balloon valvuloplasty, surgery
<i>Tricuspid Atresia</i>	Absence of tricuspid valve, leading to inadequate pulmonary blood flow	Cyanosis, dyspnea, fatigue	Prostaglandin E1, shunt placement, Fontan procedure
<i>Ebstein Anomaly</i>	Malformation of the tricuspid valve and right ventricle	Cyanosis, heart failure, arrhythmias	Prostaglandin E1, surgical

			intervention as needed
<i>Transposition of the Great Arteries (TGA)</i>	Aorta and pulmonary artery are switched	Severe cyanosis, heart failure	Prostaglandin E1, arterial switch operation

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2. Discuss the role of the following in health and diseases

a) Copper.

b) Zinc.

c) Magnesium

1. Copper:

- **Role in Health:**

- **Enzymatic Reactions:** Copper is a cofactor for several enzymes (cuproenzymes) involved in critical physiological processes, including energy production, iron metabolism, and connective tissue synthesis.
- **Neurodevelopment:** It plays a role in brain development and the functioning of the nervous system.
- **Immune Function:** Copper contributes to immune defense by supporting various cellular functions of the immune system.
- **Antioxidant Defense:** It is part of the enzyme superoxide dismutase, which helps protect cells from oxidative damage.

- **Role in Disease:**

- **Copper Deficiency:** Can lead to anemia, neutropenia, osteoporosis, and neurological disorders like peripheral neuropathy.
- **Wilson's Disease:** A genetic disorder leading to copper accumulation, causing liver disease, psychiatric symptoms, and neurological impairment.
- **Excess Copper:** Can cause liver damage and gastrointestinal disturbances.

2. Zinc:

- **Role in Health:**

- **Immune Function:** Zinc is crucial for normal development and function of cells mediating innate immunity. It also has anti-inflammatory and antioxidant properties.
- **Wound Healing:** Plays a role in cell division, cell growth, wound healing, and the breakdown of carbohydrates.
- **Protein Synthesis:** Essential for protein synthesis and helps in the proper functioning of hormones.
- **DNA Synthesis:** Involved in DNA synthesis and cell division, crucial for growth and development.

- **Role in Disease:**

- **Zinc Deficiency:** Can lead to growth retardation, delayed sexual maturation, infection susceptibility, hair loss, and impaired wound healing.
- **Acrodermatitis Enteropathica:** A rare genetic disorder causing impaired zinc absorption and severe deficiency.
- **Excessive Intake:** Can lead to gastrointestinal irritation, impaired immune function, and reduction in the level of good cholesterol.

3. Magnesium:

- **Role in Health:**

- **Bone Health:** Around 60% of body magnesium is in the bones; it plays a role in bone structure and bone health.
- **Muscle Function:** Important for muscle contraction and relaxation, including the heart muscle.
- **Nerve Function:** Plays a role in regulating neurotransmitters, which send messages throughout the brain and nervous system.
- **Energy Production:** Magnesium is a cofactor in more than 300 enzyme systems that regulate diverse biochemical reactions in the body, including protein synthesis, muscle and nerve function, blood glucose control, and blood pressure regulation.

- **Role in Disease:**

- **Magnesium Deficiency:** Can cause muscle cramps, seizures, irregular heart rhythms, and personality changes.
- **Hypomagnesemia:** Associated with osteoporosis, high blood pressure, clogged arteries, hereditary heart disease, diabetes, and stroke.
- **Excess Magnesium:** Rare and usually occurs only with supplementation. Can lead to diarrhea, nausea, and abdominal cramping. Extreme cases can cause heart problems, coma, or death.

Element	Role in Health	Role in Disease
Copper	• Cofactor in enzymatic reactions (energy production, iron metabolism, connective tissue synthesis) • Neurodevelopment and nervous system functioning • Immune function support • Part of antioxidant defense (superoxide dismutase)	Deficiency: Anemia, neutropenia, osteoporosis, neurological disorders Wilson's Disease: Copper accumulation causing liver disease, psychiatric symptoms, neurological impairment Excess: Liver damage, gastrointestinal disturbances
Zinc	• Crucial for immune function, anti-inflammatory and antioxidant properties • Involved in wound healing, cell growth, and carbohydrate breakdown • Essential for protein synthesis and hormone functioning • DNA synthesis and cell division	Deficiency: Growth retardation, delayed sexual maturation, infection susceptibility, hair loss, impaired wound healing Acrodermatitis Enteropathica: Severe deficiency due to impaired absorption Excessive Intake: Gastrointestinal irritation, impaired immune function, reduced good cholesterol levels
Magnesium	• Integral to bone health and structure • Important for muscle contraction and relaxation, including heart muscle • Regulates neurotransmitters for brain and nervous system	Deficiency: Muscle cramps, seizures, irregular heart rhythms, personality changes Hypomagnesemia: Associated with osteoporosis, high blood pressure, clogged arteries, heart disease, diabetes, stroke

	• Cofactor in over 300 enzyme systems (protein synthesis, muscle/nerve function, blood glucose control, blood pressure regulation)	Excess Magnesium: Rare, can cause diarrhea, nausea, abdominal cramping, severe cases lead to heart problems, coma, death
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3. a) Describe the evolutionary development of the hemoglobin's from embryo to adult.

- ❖ The evolutionary development of hemoglobin from the embryonic stage to adulthood is a fascinating process, reflecting the changing oxygen requirements of the human body as it develops from an embryo to a fully grown adult
- ❖ This evolutionary development of hemoglobin is a remarkable example of how the human body adapts to different stages of growth and varying oxygen requirements
- ❖ The transition from fetal to adult hemoglobin occurs shortly before and after birth, a process known as hemoglobin switching. This switch is important for adapting to the oxygen in extrauterine environment.
- ❖ It also has clinical significance, particularly in conditions like thalassemias and sickle cell disease, where understanding the dynamics of hemoglobin production can aid in treatment strategies.

1. Embryonic Hemoglobins:

- **Types:** Gower 1 ($\zeta_2\varepsilon_2$), Gower 2 ($\alpha_2\varepsilon_2$), and Portland ($\zeta_2\gamma_2$).
- **Function:** These hemoglobins are produced during the early embryonic stages. They have a high affinity for oxygen, which is crucial for the developing embryo as it extracts oxygen from the relatively low-oxygen environment of the maternal blood.
- **Timeframe:** They are predominantly present during the first 8-10 weeks of gestation.

2. Fetal Hemoglobin (HbF):

- **Composition:** $\alpha_2\gamma_2$.

- **Function:** HbF replaces embryonic hemoglobins and is the primary hemoglobin during fetal life. It has a higher affinity for oxygen than adult hemoglobin (HbA), which is advantageous for fetal oxygen transport as it facilitates the transfer of oxygen from the mother's blood (which contains HbA) to the fetal blood.
- **Timeframe:** HbF is the dominant hemoglobin from around 10 weeks of gestation until birth, and its levels start to decrease after birth.

3. **Transitional Phase:**

- **Process:** After birth, there's a transitional phase where the levels of HbF decrease, and the production of adult hemoglobin increases.
- **Significance:** This transition is crucial as the newborn now breathes air and requires a different oxygen-binding dynamics compared to the intrauterine environment.

4. **Adult Hemoglobins:**

- **Types:** HbA ($\alpha_2\beta_2$) and HbA2 ($\alpha_2\delta_2$).
- **HbA:** This is the predominant form of hemoglobin in adults, making up about 97% of the total hemoglobin. It has a lower affinity for oxygen compared to HbF, which is suitable for postnatal life where oxygen is more readily available.
- **HbA2:** It constitutes about 2-3% of the total hemoglobin in adults and plays a minor role in oxygen transport.
- **Timeframe:** The switch to adult hemoglobin is largely complete by 6 months of age, although HbF can still be present in small amounts.

5. **Genetic Regulation:**

- **Control:** The switch from fetal to adult hemoglobin is genetically controlled. The genes for
 - α -globin are located on chromosome 16,
 - β -globin, γ -globin (fetal), δ -globin (adult), and ϵ -globin (embryonic) are located on chromosome 11.
- **Regulatory Mechanisms:** Various transcription factors and regulatory proteins are involved in the suppression of fetal hemoglobin production and the activation of adult hemoglobin production.

Stage	Hemoglobin Type	Composition	Characteristics
Embryonic	Gower 1 ($\zeta 2\epsilon 2$)	Two zeta and two epsilon chains	Predominant in early embryonic life, high oxygen affinity
	Gower 2 ($\alpha 2\epsilon 2$)	Two alpha and two epsilon chains	Appears slightly later in embryonic development
	Portland ($\zeta 2\gamma 2$)	Two zeta and two gamma chains	Found in early embryonic life, high oxygen affinity
Fetal	Hb F ($\alpha 2\gamma 2$)	Two alpha and two gamma chains	Predominant in fetal life, higher oxygen affinity than adult hemoglobin, facilitates oxygen transfer from mother to fetus
Adult	Hb A ($\alpha 2\beta 2$)	Two alpha and two beta chains	Main adult hemoglobin, lower oxygen affinity compared to fetal hemoglobin, allows for efficient oxygen release to tissues
	Hb A2 ($\alpha 2\delta 2$)	Two alpha and two delta chains	Minor adult hemoglobin, slightly higher oxygen affinity than Hb A

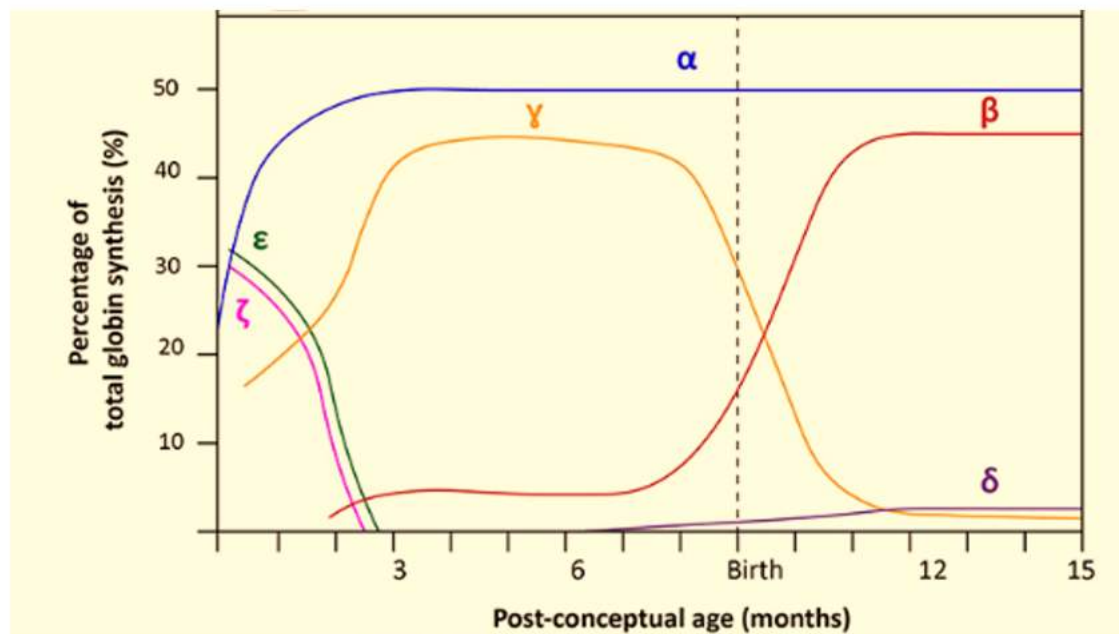
Notes:

- **Embryonic Hemoglobins:** These are the first to appear and are characterized by their high oxygen affinity, which is crucial for the early stages of embryonic development.
- **Fetal Hemoglobin (Hb F):** It has a higher affinity for oxygen than adult hemoglobin, which is essential for the fetus to extract oxygen from the maternal blood supply.
- **Adult Hemoglobins (Hb A and Hb A2):** These have lower oxygen affinity, which is more suited for oxygen delivery to the tissues under normal physiological conditions.

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3. b) Describe the relationship among the various hemoglobin's in first year of life and their implication in diagnosing hereditary hemoglobinopathies.

- ❖ Understanding the dynamics of hemoglobin types during the first year of life is essential for diagnosing and managing hereditary hemoglobinopathies.
- ❖ It helps in identifying carriers of genetic mutations and in planning appropriate interventions to manage these conditions effectively.



1. **Hemoglobin F (Hb F):** At birth, Hb F ($\alpha_2\gamma_2$) is the predominant hemoglobin, constituting about 70-95% of the total hemoglobin. Hb F has a higher affinity for oxygen than adult hemoglobin, which is essential for fetal life.
2. **Hemoglobin A (Hb A):** This is the primary adult hemoglobin ($\alpha_2\beta_2$). Its synthesis begins before birth but increases significantly after birth. By 6 months of age, Hb A becomes the predominant hemoglobin.
3. **Hemoglobin A2 (Hb A2):** This is a minor adult hemoglobin ($\alpha_2\delta_2$). Its levels are usually low at birth and gradually increase to adult levels (about 2-3% of total hemoglobin) by the end of the first year.
4. **Hemoglobin Switching:** The transition from Hb F to Hb A, known as hemoglobin switching, is a complex process regulated by various genetic factors. This switch is essential for adapting to the oxygen environment outside the womb.
5. **Implications in Diagnosing Hereditary Hemoglobinopathies:**

- **Thalassemias:** These are characterized by reduced synthesis of one of the globin chains. In beta-thalassemia, there is a reduced synthesis of beta chains, leading to relatively high levels of Hb F and Hb A₂. In alpha-thalassemia, the production of alpha chains is affected, which can alter the ratios of different hemoglobins.
 - **Sickle Cell Disease:** This is caused by a mutation in the beta-globin gene. Newborn screening can detect the presence of hemoglobin S (Hb S). The disease's severity can be modulated by the proportion of Hb F.
 - **Other Hemoglobin Variants:** Various other abnormal hemoglobins (e.g., Hb C, Hb E) can be identified through newborn screening and subsequent hemoglobin electrophoresis or HPLC.
6. **Newborn Screening and Follow-up:** Newborn screening for hemoglobinopathies is crucial for early diagnosis. Follow-up testing in the first year of life, including quantitative analysis of Hb F, Hb A, and Hb A₂, can help in the definitive diagnosis and management of these conditions.

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4. a) Illustrate the pathophysiology of R-L shunt.

The pathophysiology of a right-to-left (R-L) shunt in the heart involves the abnormal flow of blood from the right side of the heart to the left side, bypassing the lungs. This can occur due to various congenital heart defects.

Normal Circulation: In a healthy heart, oxygen-poor blood returns from the body to the right atrium, flows to the right ventricle, and then is pumped to the lungs to get oxygenated. The oxygen-rich blood returns to the left atrium, flows into the left ventricle, and is then pumped out to the body.

1. **R-L Shunt Mechanism:**

- **Defects Leading to R-L Shunt:** Conditions such as Tetralogy of Fallot, Transposition of the Great Arteries, Truncus Arteriosus, Total Anomalous Pulmonary Venous Return, or a large Ventricular Septal Defect (VSD) with pulmonary hypertension can result in a R-L shunt.
- **Abnormal Blood Flow:** In these conditions, blood flows from the right side of the heart to the left side, bypassing the lungs. This happens due to either an anatomical defect (like a hole between the heart chambers)

or physiological conditions (like pulmonary hypertension) that reverse the normal pressure gradient.

2. **Consequences of R-L Shunt:**

- **Reduced Oxygenation:** Since the blood bypasses the lungs, it does not get adequately oxygenated. This leads to lower oxygen levels in the systemic circulation.
- **Cyanosis:** The most noticeable symptom of a R-L shunt is cyanosis, which is a bluish coloration of the skin and mucous membranes due to the increased amount of deoxygenated blood in the systemic circulation.
- **Hypoxemia:** Reduced oxygen levels in the blood can lead to hypoxemia, affecting various organ systems.
- **Polycythemia:** The body may respond to chronic hypoxemia by increasing red blood cell production (polycythemia), which can lead to increased blood viscosity and risk of thrombosis.
- **Clubbing:** Chronic hypoxemia can also lead to clubbing of the fingers and toes.

3. **Complications:**

- **Paradoxical Embolism:** If a clot forms in the venous system, it can cross over to the systemic circulation through the shunt, potentially causing a stroke or other systemic embolic events.
- **Eisenmenger Syndrome:** In some cases, long-standing left-to-right shunts can lead to pulmonary hypertension, which eventually reverses the shunt to a right-to-left direction.

4. **Diagnosis and Management:**

- **Echocardiography:** This is the primary diagnostic tool to identify structural defects causing the shunt.
- **Oxygen Saturation Monitoring:** To assess the extent of hypoxemia.
- **Surgical Correction:** Most R-L shunts require surgical intervention to correct the underlying defect and normalize blood flow.

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5. a) Development of diaphragm, trachea and esophagus and types of TOF with diagrams.

Embryology and Development of Esophagus

- **Foregut Formation:** The esophagus originates from the dorsal part of the foregut tube during the 4th week of gestation.
- **Endodermal Layer:** The inner lining of the esophagus is derived from endoderm.
- **Epithelial Occlusion and Recanalization:** Rapid proliferation of epithelial cells leads to temporary occlusion of the lumen, which recanalizes by the 10th week.
- **Muscularis Externa:** Mesenchymal cells differentiate into smooth muscle cells, forming the muscularis externa.
- **Stratified Squamous Epithelium:** Differentiation of the epithelium into a stratified squamous type occurs later in development.
- **Glandular Tissue:** Submucosal and esophageal glands develop from endodermal outgrowths.

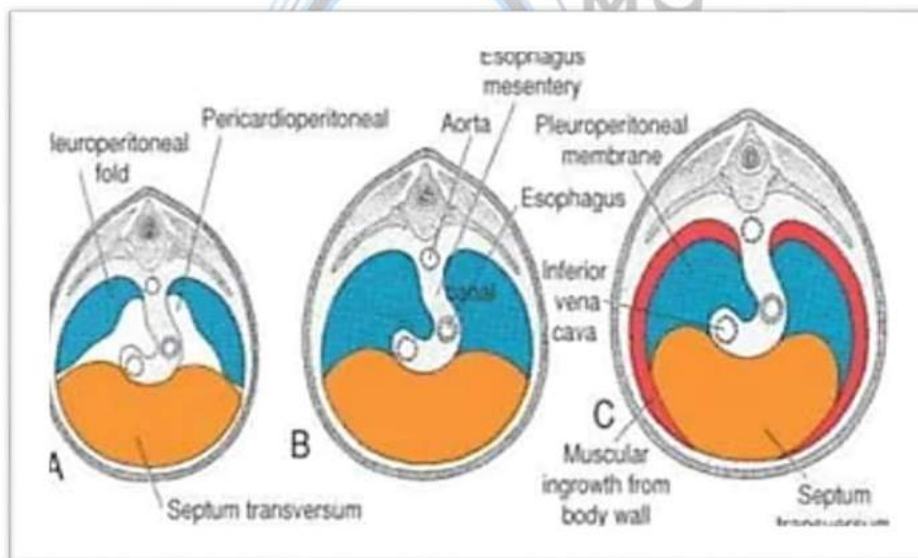
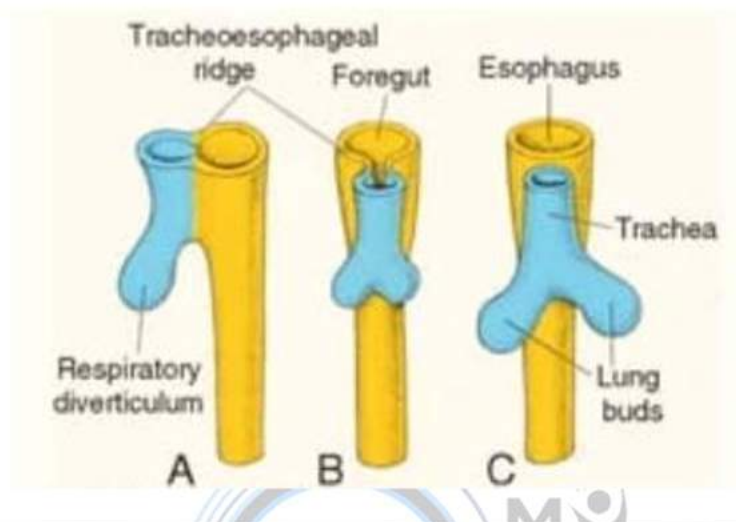
Embryology and Development of Trachea

- **Foregut Division:** The trachea arises from the ventral part of the foregut, separated by the tracheoesophageal septum.
- **Lung Bud Formation:** The lung buds emerge from the ventral wall of the primitive trachea during the 4th week.
- **Tracheoesophageal Fistula:** Failure of the tracheoesophageal septum to divide the foregut properly can lead to this congenital anomaly.
- **Cartilage and Muscle Formation:** Mesenchymal cells surrounding the endodermal tube differentiate into tracheal cartilage and smooth muscle.
- **Bronchial Tree:** The lung buds further divide to form the bronchial tree, under the influence of various growth factors.

Embryology and Development of Diaphragm

- **Septum Transversum:** Initially forms the central tendon; originates from the mesoderm in the cervical region.
- **Pleuroperitoneal Folds:** These structures close the pericardioperitoneal canals, separating the thoracic and abdominal cavities.

- **Muscular Component:** Myoblasts from the cervical somites migrate along the phrenic nerves to form the muscular diaphragm.
- **Descent of Diaphragm:** The diaphragm descends to its final thoracic position, allowing for lung expansion.
- **Innervation:** The phrenic nerve, derived from the C3-C5 spinal nerves, innervates the diaphragm, enabling its contraction and thus respiration.



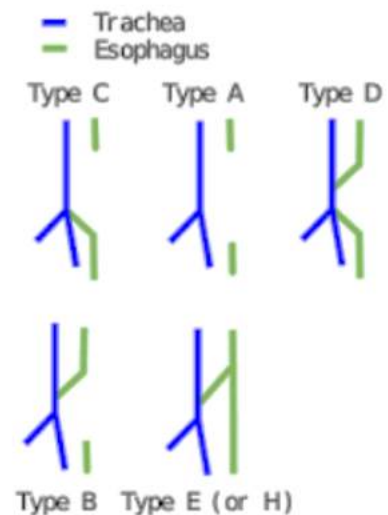
Tracheoesophageal Fistula and Esophageal atresia

Esophageal Atresia (EA)

- Embryological Basis: Occurs due to the failure of the tracheoesophageal septum to properly separate the foregut into the trachea and esophagus.

- Types of EA:**

- Type A: Isolated EA without TEF
- Type B: EA with proximal TEF
- Type C: EA with distal TEF (most common)
- Type D: EA with both proximal and distal TEF
- Type E: H-type, no atresia but TEF present



- Clinical Manifestations:**

- Polyhydramnios in utero
- Frothy bubbles in the mouth and nose
- Coughing, choking, and cyanosis during feeding

- Diagnosis:**

- Prenatal ultrasound may show polyhydramnios
- Failure to pass a nasogastric tube into the stomach
- Chest X-ray and fluoroscopic swallow study

- Management:**

- Surgical correction is the definitive treatment
- Preoperative stabilization with gastric decompression and ventilatory support

Tracheoesophageal Fistula (TEF)

- Embryological Basis: Abnormal persistence of the tracheoesophageal groove leads to the formation of a fistulous connection.

- Types of TEF:**

- Proximal TEF: Connects the upper esophagus to the trachea
- Distal TEF: Connects the lower esophagus to the trachea

- H-type: No atresia but a fistula exists between the trachea and esophagus
- **Clinical Manifestations:**
 - Recurrent pneumonia
 - Abdominal distension
 - Aspiration during feeding
- **Diagnosis:**
 - Contrast-enhanced fluoroscopic study
 - Bronchoscopy for direct visualization
 - MRI or CT scans for complex cases
- **Management:**
 - Surgical ligation of the fistula
 - Esophageal reconstruction in cases of atresia
 - Postoperative care includes antibiotic prophylaxis and nutritional support

5. b) Briefly describe the important attributes of various patterns of genetic inheritance

Patterns of genetic inheritance:

1. **Autosomal Dominant Inheritance:**

- **One Affected Parent:** Typically, one parent is affected and has a 50% chance of passing the gene to each child.
- **Both Genders Affected Equally:** This pattern affects males and females equally.
- **No Generation Skipping:** The trait often appears in every generation.
- **Variable Expressivity:** Severity of the trait can vary among individuals.
- **Examples:** Huntington's disease, Marfan syndrome.

2. **Autosomal Recessive Inheritance:**

- **Carriers:** Parents are usually carriers (having one normal and one affected gene) and are typically unaffected.
- **25% Risk for Each Child:** Each child has a 25% chance of being affected, a 50% chance of being a carrier, and a 25% chance of being unaffected.
- **Both Genders Affected Equally:** Males and females are equally likely to be affected.
- **Can Skip Generations:** The trait can skip generations if the gene is carried silently.
- **Examples:** Cystic fibrosis, Sickle cell anemia.

3. **X-linked Recessive Inheritance:**

- **Mostly Affects Males:** Males are more frequently affected as they have only one X chromosome.
- **Carrier Mothers:** Mothers are usually carriers and can pass the gene to their sons (50% chance) and daughters (50% chance of being carriers).
- **No Father-to-Son Transmission:** The gene is not passed from father to son, as males contribute a Y chromosome to their sons.
- **Examples:** Hemophilia, Duchenne muscular dystrophy.

4. **X-linked Dominant Inheritance:**

- **Both Genders Affected:** Both males and females can be affected, but the disease may be more severe in males.
- **Affected Fathers:** All daughters of an affected father will inherit the condition, but none of his sons will.
- **Carrier Mothers:** Affected mothers have a 50% chance of passing the condition to each child, regardless of gender.
- **Examples:** Fragile X syndrome, Rett syndrome.

5. **Mitochondrial Inheritance:**

- **Maternal Transmission:** Mitochondrial genes are passed exclusively by mothers to all their children.
- **Both Genders Affected:** Both males and females can be affected, but only females pass it on.

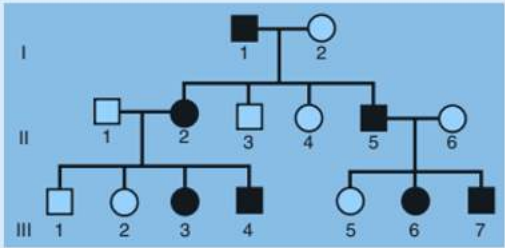
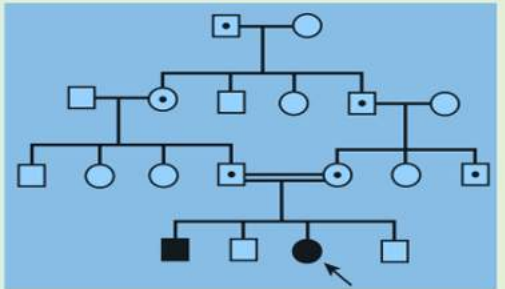
- **Variable Expression:** Severity can vary widely, even within the same family.
- **Examples:** Leber's hereditary optic neuropathy, Mitochondrial myopathy.

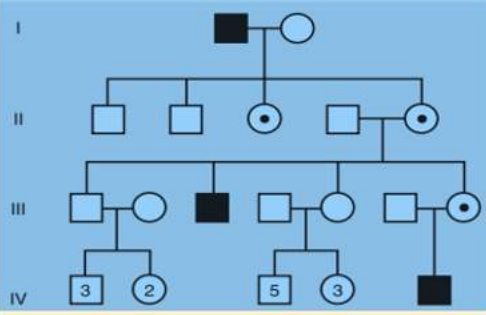
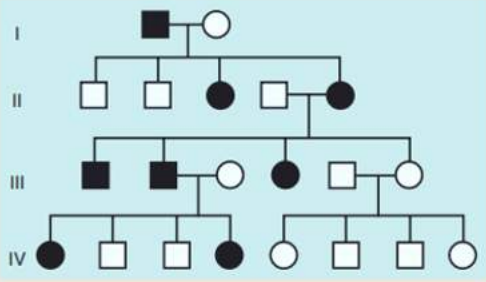
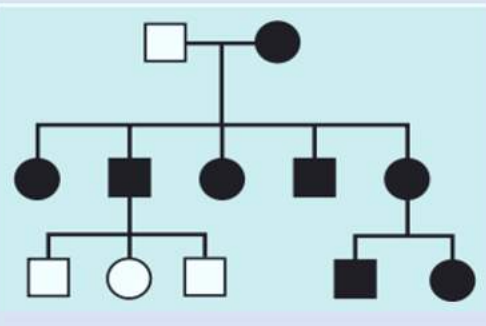
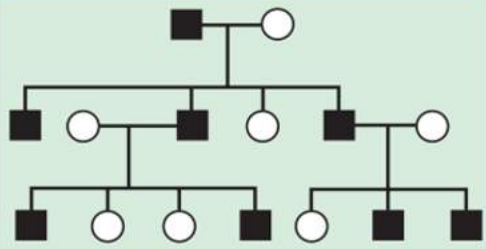
6. **Y-linked Inheritance:**

- **Only Affects Males:** Only males have Y chromosomes, so this pattern is seen only in males.
- **Passed from Father to Son:** Traits are passed directly from father to all sons.
- **Examples:** Y-chromosome infertility, some cases of male pattern baldness.

Each pattern of inheritance has unique characteristics that influence how genetic conditions and traits are passed through families.

Understanding these patterns is crucial for genetic Counselling, diagnosis, and management of hereditary conditions.

Pattern of Inheritance/ Pedigree	Key Attributes	Gender Affected	Generation Skipping	Examples
Autosomal Dominant 	One affected parent; 50% chance of passing the gene; variable expressivity	Both	No	Huntington's disease, Marfan syndrome
Autosomal Recessive 	Parents usually carriers; 25% risk for each child; can skip generations	Both	Yes	Cystic fibrosis, Sickle cell anemia

X-linked Recessive 	Mostly affects males; carrier mothers; no father-to-son transmission	Mostly Males	Possible	Hemophilia, Duchenne muscular dystrophy
X-linked Dominant 	Both genders affected; affected fathers pass to all daughters; carrier mothers have 50% chance of passing to each child	Both	No	Fragile X syndrome, Rett syndrome
Mitochondrial 	Maternal transmission; variable expression; affects both genders, but only females pass it on	Both	No	Leber's hereditary optic neuropathy, Mitochondrial myopathy
Y-linked 	Only affects males; passed from father to all sons	Only Males	No	Y-chromosome infertility, Male pattern baldness

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6. List the various study designs for biomedical research with one suitable example for each

Study Design	Description	Example
<i>Randomized Controlled Trial (RCT)</i>	A study where participants are randomly assigned to a treatment or control group to compare outcomes.	A study comparing a new drug versus a placebo in reducing blood pressure in hypertensive patients.
<i>Cohort Study</i>	Observational study where a group (cohort) is followed over time to observe outcomes.	A long-term study following smokers and non-smokers to compare lung cancer incidence.
<i>Case-Control Study</i>	A retrospective study comparing individuals with a specific condition (cases) to those without (controls).	Comparing past exposure to risk factors between patients with a specific disease and those without.
<i>Cross-Sectional Study</i>	Observes a specific population at a single point in time to assess prevalence and relationships.	A survey assessing obesity prevalence and associated lifestyle factors in adults.
<i>Case Series</i>	A descriptive study detailing characteristics of a group of patients with a common condition.	A report on patients treated with a novel therapy for a rare skin disorder.
<i>Case Report</i>	Detailed report of the symptoms, diagnosis, treatment, and follow-up of an individual patient.	A detailed presentation of a patient with an unusual presentation of a common disease.
<i>Systematic Review</i>	A comprehensive review and synthesis of research studies on a particular topic.	Review synthesizing multiple studies on exercise effectiveness in reducing depression.
<i>Meta-Analysis</i>	Combines data from multiple studies to derive conclusions about a specific research question.	Analysis combining several studies to determine overall effectiveness of antiviral drugs for influenza.
<i>Ecological Study</i>	Study of groups or populations, looking at the correlation between	Examining the correlation between national dietary habits and heart disease prevalence.

	exposure and outcome at the group level.	
<i>Controlled Before-and-After Study</i>	Observes outcomes before and after a specific intervention in a controlled environment.	Assessing the impact of a new traffic law on road accident rates before and after its implementation.
<i>Quasi-Experimental Study</i>	Resembles an experimental study but lacks random assignment to treatment or control.	Investigating the effects of a new teaching method on student performance in schools that adopted it versus those that did not.
<i>Longitudinal Study</i>	Follows the same subjects over a long period to observe changes or outcomes.	Tracking the mental health of individuals over several decades.
<i>Narrative Review</i>	Provides a broad overview of a topic, based on a collection of studies and literature.	Overview of the literature on the mechanisms of action of a specific class of antibiotics.

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7. a) Briefly describe the indicators of child health

The Government of India uses various indicators to monitor and assess child health. These indicators are crucial for understanding the health status of children across the country and for guiding policy and programmatic interventions.

These indicators are used in various national health surveys and reports, such as the National Family Health Survey (NFHS), to monitor trends and guide health policies and programs in India.

1. **Infant Mortality Rate (IMR):** This measures the number of deaths of infants under one year of age per 1,000 live births. It's a critical indicator of the overall health status of a population, reflecting the effectiveness of maternal and child health services.
2. **Under-Five Mortality Rate (U5MR):** This represents the probability of dying before the age of five per 1,000 live births. It's an important measure of child survival and overall development.

3. **Neonatal Mortality Rate (NMR):** This is the number of deaths during the first 28 completed days of life per 1,000 live births. It indicates the quality of antenatal care, childbirth, and postnatal care services.
4. **Immunization Coverage:** This assesses the percentage of children who have received essential vaccines (like DPT, BCG, Measles, Polio) by a certain age. High immunization coverage is crucial for preventing childhood diseases.
5. **Nutritional Status Indicators:** These include the prevalence of underweight, stunting (low height-for-age), and wasting (low weight-for-height) among children. These indicators reflect the nutritional health and food security of children.
6. **Breastfeeding Practices:** Early initiation of breastfeeding and exclusive breastfeeding for the first six months are important indicators of child health, impacting infant survival and long-term health.
7. **Anaemia Prevalence:** The proportion of children with anaemia is an indicator of nutritional deficiencies and impacts cognitive and physical development.
8. **Birth Weight:** The percentage of newborns with low birth weight (less than 2.5 kg) is a crucial indicator of maternal health, nutrition, and prenatal care.
9. **Institutional Deliveries:** The rate of births occurring in healthcare facilities indicates access to and utilization of maternal health services.
10. **Sanitation and Hygiene:** Access to clean water and sanitation facilities, and practices like handwashing, are important for preventing infections and maintaining child health.
11. **Education and Literacy Rates:** Though indirectly related, education and literacy rates of mothers significantly impact child health outcomes.
12. **Child Disease Prevalence:** Data on the prevalence of common childhood diseases like diarrhea, respiratory infections, and others are used to assess health status and healthcare needs.

Child Health Indicators in India (as of 2022)

Indicator	Description	Latest Data (as of 2022)
Stunting (height-for-age)	Prevalence in children under 5 years	Approximately 35%
Wasting (weight-for-height)	Prevalence in children under 5 years	Around 17%

Underweight (weight-for-age)	Prevalence in children under 5 years	Nearly 33%
Immunization (BCG)	Percentage of children vaccinated	About 91%
Immunization (DPT)	Percentage of children receiving three doses	Approximately 78%
Immunization (Polio)	Percentage of children receiving three doses	Around 76%
Immunization (Measles)	Percentage of children vaccinated	About 81%
Infant Mortality Rate (IMR)	Number of deaths per 1,000 live births	Approximately 30 deaths/1,000 live births
Under-5 Mortality Rate	Number of deaths per 1,000 live births	Around 36 deaths/1,000 live births
Exclusive Breastfeeding	Percentage of infants 0-6 months exclusively breastfed	Nearly 55%
Access to Improved Drinking Water	Percentage of population with access	Over 90%
Access to Improved Sanitation Facilities	Percentage of population with access	Approximately 60%
Diarrheal Diseases (Incidence)	Incidence among children under 5 years	High, specific data varies regionally
Respiratory Infections (Incidence)	Incidence among children under 5 years	Common, specific data varies
Antenatal Care (ANC)	Percentage of women receiving ANC	About 51% receiving full ANC
Institutional Deliveries	Percentage of births in health facilities	Over 80%

- **Data Variability:** Not that the data presented here are approximations and may vary slightly based on different sources and updates.
- **Regional Differences:** There are significant regional variations in these indicators across different states in India.
- **Source Reliability:** These figures are based on reports from organizations like UNICEF, WHO, and the Government of India.

- **Recent Updates:** For the most recent and specific data, visit the latest reports and databases from relevant health organizations from their official websites before your exams.

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7. b. List the major programmatic interventions to reduce the IMR.

The following interventions represent a multifaceted approach, combining healthcare service delivery, community engagement, and policy initiatives to address the complex determinants of infant mortality in India.

- **National Health Mission (NHM):**
 - Aims to establish a fully functional, community-owned, decentralized health delivery system.
 - Includes two sub-missions: National Rural Health Mission (NRHM) and National Urban Health Mission (NUHM).
 - Focuses on strengthening health systems, improving access to rural healthcare, and ensuring availability of free services to the vulnerable population.
- **Integrated Child Development Services (ICDS) Scheme:**
 - Targets early childhood care and development.
 - Provides a package of services including supplementary nutrition, immunization, health check-up, referral services, pre-school non-formal education, and nutrition & health education.
 - Aims to reduce infant and young child undernutrition and mortality.
- **Janani Suraksha Yojana (JSY):**
 - A safe motherhood intervention under the NHM.
 - Promotes institutional delivery among poor pregnant women.
 - Offers cash assistance with antenatal care during the pregnancy period and institutional care during delivery and immediate postpartum period.
- **Janani Shishu Suraksha Karyakram (JSSK):**
 - Ensures free and cashless services to pregnant women including normal deliveries and caesarean operations.

- Extends to sick newborns up to 30 days after birth.
- Aims to reduce out-of-pocket expenses for both pregnant women and sick newborns.
- ***Rashtriya Bal Swasthya Karyakram (RBSK):***
 - An initiative aimed at screening children from birth to 18 years for 4 'D's - Defects at birth, Diseases, Deficiencies, and Development delays including disabilities.
 - Includes early intervention and management of identified cases.
- ***Mission Indradhanush:***
 - Aims to immunize all children under the age of 2 years, as well as all pregnant women, against seven vaccine-preventable diseases.
 - Diseases targeted include diphtheria, whooping cough, tetanus, poliomyelitis, tuberculosis, measles, and hepatitis B.
- ***Pradhan Mantri Matru Vandana Yojana (PMMVY):***
 - A maternity benefit program that provides a cash incentive to pregnant and lactating mothers for the first living child.
 - Encourages women to seek pre and post-natal care and immunization for their children.
- ***ASHA (Accredited Social Health Activist) Program:***
 - Part of the NHM, ASHAs are community health workers instituted by the Ministry of Health and Family Welfare.
 - Act as a bridge between the community and the health system, facilitating access to healthcare services, especially for rural and marginalized populations.
- ***Facility-Based Newborn Care (FBNC):***
 - Establishes Newborn Care Corners (NBCCs) at every point of child delivery, Newborn Stabilization Units (NBSUs) for sick newborns, and Special Newborn Care Units (SNCUs) for critical care.
 - Aims to reduce neonatal mortality by providing comprehensive care for newborns at the community and facility level.
- ***Home-Based Newborn Care (HBNC):***

- Involves health workers providing care at the household level to newborns and mothers.
- Focuses on essential newborn care practices, identification, and referral of sick newborns.
- **Strengthening of Maternal and Neonatal Health Services:**
 - Enhances the quality of healthcare during pregnancy, childbirth, and postnatal periods.
 - Involves training of health personnel in basic and comprehensive obstetric care.
- **Public-Private Partnerships (PPPs):**
 - Engages with private sector providers for delivering reproductive and child health services.
 - Aims to supplement the efforts of the public health system in areas where it lacks reach or capacity.

Programmatic Intervention	Description	Contribution to Reducing IMR
National Health Mission (NHM)	Focuses on strengthening health systems and improving access to healthcare, especially in rural areas.	Enhanced healthcare infrastructure and accessibility have led to better maternal and child health services, thus reducing IMR.
Integrated Child Development Services (ICDS) Scheme	Provides services like supplementary nutrition, health check-ups, and immunization.	Improved nutrition and healthcare in early childhood have contributed to lower child mortality rates.
Janani Suraksha Yojana (JSY)	Promotes institutional deliveries among poor pregnant women with cash incentives.	Increased institutional deliveries have led to better maternal and newborn care, reducing neonatal and infant deaths.
Janani Shishu Suraksha Karyakram (JSSK)	Offers free and cashless services to pregnant women and sick newborns.	Reduced the financial burden and improved access to healthcare for mothers and newborns, leading to lower mortality rates.

<i>Rashtriya Bal Swasthya Karyakram (RBSK)</i>	Aims at early detection and management of common childhood illnesses.	Early intervention in childhood diseases has helped in reducing the mortality associated with these conditions.
<i>Mission Indradhanush</i>	Targets immunization against seven vaccine-preventable diseases.	Enhanced immunization coverage has led to a decrease in infant deaths due to preventable diseases.
<i>Pradhan Mantri Matru Vandana Yojana (PMMVY)</i>	Provides cash incentives to pregnant and lactating mothers.	Encourages healthcare utilization during pregnancy and postnatal period, improving maternal and child health outcomes.
<i>ASHA Program</i>	Employs community health workers to facilitate healthcare access.	Improved community-level health education and services have contributed to better neonatal and infant care.
<i>Facility-Based Newborn Care (FBNC)</i>	Establishes various levels of newborn care units in healthcare facilities.	Enhanced care for newborns, especially those with complications, has significantly reduced neonatal mortality.
<i>Home-Based Newborn Care (HBNC)</i>	Involves health workers providing care at the household level.	Improved newborn care practices at home and early identification of complications have lowered infant deaths.
<i>Strengthening of Maternal and Neonatal Health Services</i>	Focuses on improving the quality of healthcare during pregnancy, childbirth, and the postnatal period.	Better quality of maternal healthcare has led to healthier births and reduced infant mortality.
<i>Public-Private Partnerships (PPPs)</i>	Engages private sector in delivering reproductive and child health services.	Supplementing public health efforts, PPPs have expanded the reach and quality of maternal and child health services.

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8.a) a) Chi Square test (illustrate with an example a setting where it should be used)

The Chi-Square test is a robust statistical tool for assessing the relationship between two categorical variables.

In medical research, the Chi-Square test is applied under specific conditions, primarily when dealing with categorical data. Here are the key scenarios where this test is relevant:

1. Testing Association between Two Categorical Variables:

- To determine if there is a significant association or relationship between two categorical variables.
- Example: Investigating the relationship between smoking status (smoker/non-smoker) and the presence of lung disease (yes/no).

2. Comparing Observed Frequencies with Expected Frequencies:

- To test if the observed frequencies in each category significantly differ from what would be expected under a null hypothesis.
- Example: Comparing the actual distribution of blood types in a sample with the expected distribution based on known population frequencies.

3. Evaluating the Effectiveness of a Treatment or Intervention:

- To assess the efficacy of a new treatment or intervention in comparison to a control or standard treatment.
- Example: Evaluating the effectiveness of a new vaccine by comparing infection rates in vaccinated vs. unvaccinated groups.

4. Testing for Homogeneity:

- To determine if different populations or groups have similar distributions of a categorical variable.
- Example: Comparing the distribution of a health condition across different demographic groups to see if it's consistent.

5. Goodness-of-Fit Test:

- To see if a sample data fits a distribution from a certain population.
- Example: Testing if the incidence of a particular disease follows an expected epidemiological distribution.

6. Contingency Table Analysis:

- To analyze and interpret data presented in a contingency table (cross-tabulation) involving two or more categorical variables.
- Example: Analyzing a 2x2 table showing the presence or absence of a disease in relation to exposure to a risk factor.

7. *Post-Hoc Analysis in ANOVA:*

- When ANOVA indicates significant differences, Chi-Square tests can be used for post-hoc analysis to identify where those differences lie among categorical variables.
- Example: After finding a significant effect of a drug on disease symptoms, using Chi-Square to analyze which specific symptoms were most affected.

Conditions for Validity of the Chi-Square Test:

- **Sample Size:** Each cell in the contingency table should have an expected frequency of 5 or more for the test to be valid.
- **Independence:** Observations should be independent of each other.
- **Random Sampling:** Data should be collected through a random process.

In pediatrics, it can be particularly useful for exploring associations between different treatment modalities and outcomes, as illustrated in the example.

- **Conceptual Overview**
 - **Purpose:** The Chi-Square test is used to examine the association between two categorical variables.
 - **Assumptions:**
 - Observations are independent.
 - Sample size is sufficiently large (usually, at least 5 observations in each cell of the contingency table).
- **Example**
 - **Research Question:** Is there an association between the type of feeding (breastfed vs. formula-fed) and the incidence of atopic dermatitis in infants?
- **Data Collection**

- **Sample Size:** 200 infants
- **Variables:**
 - Type of Feeding: Breastfed, Formula-fed
 - Incidence of Atopic Dermatitis: Yes, No
- **Contingency Table**

	Atopic Dermatitis: Yes	Atopic Dermatitis: No	Row Total
Breastfed	20	80	100
Formula-fed	40	60	100
Column Total	60	140	200

- **Chi-Square Calculation**
 - **Formula:** $\chi^2 = \sum E(O-E)^2/E$
 - O = Observed Frequency
 - E = Expected Frequency
 - **Expected Frequency for Breastfed & Atopic Dermatitis: Yes:**
 - $(100 \times 60)/200 = 30$
 - **Chi-Square for this cell:** $(20-30)^2/30 = 100/30 = 3.3330$
 - **Repeat for all cells and sum up:** $\chi^2 = 3.33 + \dots \chi^2 = 3.33 + \dots$
- **Degrees of Freedom**
 - $df = (\text{Number of Rows} - 1) \times (\text{Number of Columns} - 1)$
 - $df = (2-1) \times (2-1) = 1$
- **Significance Level**
 - Usually set at 0.05
- **Interpretation**
 - **Critical Value:** Obtain from Chi-Square distribution table (for $df=1$ and $\alpha=0.05$, critical value is 3.841)
 - **Decision Rule:** If χ^2 calculated > Critical Value, reject the null hypothesis.

- **Conclusion:** If χ^2 calculated is greater than 3.841, there is a significant association between the type of feeding and the incidence of atopic dermatitis in infants.

Another simple example:

Scenario: A pharmaceutical company has developed a new vaccine for a particular disease and wants to test its efficacy. The study involves two groups: one receiving the vaccine (treatment group) and the other receiving a placebo (control group). After a certain period, the number of individuals who developed the disease and those who did not are recorded in each group.

Data Collection:

- **Group 1 (Vaccine):** 100 individuals
 - Developed Disease: 10
 - Did Not Develop Disease: 90
- **Group 2 (Placebo):** 100 individuals
 - Developed Disease: 30
 - Did Not Develop Disease: 70

Objective: To determine if there is a statistically significant difference in the proportion of individuals who develop the disease between the vaccine group and the placebo group.

Application of Chi-Square Test

1. Setting Up the Hypotheses:

- **Null Hypothesis (H_0):** There is no association between the vaccine and the incidence of the disease. (The vaccine is not effective)
- **Alternative Hypothesis (H_1):** There is an association between the vaccine and the incidence of the disease. (The vaccine is effective)

2. Constructing the Contingency Table:

	Developed Disease	Did Not Develop Disease	Total
Vaccine Group	10	90	100

Placebo Group	30	70	100
Total	40	160	200

3. **Calculating the Chi-Square Statistic:**

- The formula involves calculating the expected counts under the null hypothesis, comparing them with the observed counts, and summing their squared differences divided by the expected counts.
- The calculation would yield a Chi-Square statistic (χ^2 value).

4. **Determining the Significance:**

- Compare the calculated χ^2 value with the critical value from the Chi-Square distribution table at a chosen significance level (commonly 0.05).
- If the calculated χ^2 is greater than the critical value, reject the null hypothesis.

5. **Conclusion:**

- If the null hypothesis is rejected, it suggests that the vaccine has a significant effect on the incidence of the disease.
- If the null hypothesis is not rejected, it suggests that there is no statistical evidence to conclude that the vaccine is effective.

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8.b Quality Improvement Initiatives in healthcare (illustrate with an example).

Quality Improvement (QI) initiatives in healthcare are essential for enhancing patient care, optimizing resource utilization, and improving overall health outcomes. It involves the **PDSA** cycle.

These initiatives are particularly crucial in pediatric care, where the focus is on the health and well-being of children.

Example: Improving Asthma Management in a Pediatric Population

Background:

- ❖ A pediatric clinic identifies a high rate of emergency room visits and hospitalizations among its pediatric asthma patients. This situation indicates a need for better asthma management to improve patient outcomes and reduce healthcare costs.
- ❖ This QI initiative in a pediatric setting demonstrates how a systematic, data-informed approach can significantly improve the management of a chronic condition like asthma in children
- ❖ By focusing on education, personalized care plans, and regular follow-up, the clinic aims to enhance asthma control, thereby reducing emergency interventions and improving the quality of life for pediatric patients
- ❖ This example highlights the importance of involving patients and families in care, the use of data for informed decision-making, and the need for ongoing evaluation and adaptation in quality improvement efforts.

Quality Improvement Initiative:

1. **Problem Identification and Goal Setting:**

- Problem: Elevated rates of emergency room visits and hospitalizations due to asthma exacerbations in children.
- Goal: Reduce emergency visits and hospitalizations by 25% within 12 months.

2. **Formation of a Multidisciplinary Team:**

- Team includes pediatricians, respiratory therapists, nurses, a QI specialist, and a patient education coordinator.
- Each member plays a role in intervention planning, implementation, and evaluation.

3. **Data Collection and Analysis:**

- Review medical records to identify patterns in asthma exacerbations.
- Identify potential contributing factors, such as poor medication adherence, lack of asthma action plans, or environmental triggers.

4. **Development of Interventions:**

- Create individualized asthma action plans for each patient.

- Implement a structured asthma education program for patients and families, focusing on medication use, trigger avoidance, and symptom monitoring.
- Enhance routine follow-up care, including regular asthma control assessments.

5. **Pilot Testing:**

- Test the interventions with a small group of patients.
- Gather feedback from patients, families, and staff to refine the approach.

6. **Full-Scale Implementation:**

- Expand the program to all pediatric asthma patients in the clinic.
- Train all relevant staff on the new protocols and educational materials.

7. **Monitoring and Evaluation:**

- Continuously track emergency room visits and hospitalization rates among the patient cohort.
- Collect feedback from patients and families regarding the educational program and asthma management.

8. **Outcome Assessment and Iteration:**

- After 12 months, assess the impact on emergency visits and hospitalizations.
- If the goal is achieved, consider adopting similar strategies for other chronic pediatric conditions.
- If not, analyze the data to identify barriers and modify the interventions accordingly.

Key Components of the Initiative:

- **Patient and Family Education:** Empowering patients and families with knowledge and skills for effective asthma management.
- **Personalized Care Plans:** Developing individualized asthma action plans tailored to each child's needs.
- **Data-Driven Approach:** Utilizing patient data to identify needs and measure the impact of interventions.

- **Continuous Improvement:** Regular evaluation and adaptation of strategies based on feedback and outcomes.

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9. Discuss salient features of management of children with tuberculosis as per National Tuberculosis Elimination Programme (NTEP).

The management of children with tuberculosis (TB) under the National Tuberculosis Elimination Programme (NTEP) in India involves a comprehensive approach that addresses the unique challenges and needs of pediatric TB patients.

The NTEP, previously known as the Revised National Tuberculosis Control Programme (RNTCP), has specific guidelines and strategies for the diagnosis, treatment, and follow-up of TB in children. Here are the salient features:

1. Early and Accurate Diagnosis:

- Emphasis on early detection of TB in children, which is challenging due to the paucibacillary nature of the disease and difficulties in obtaining sputum samples.
- Use of various diagnostic methods including clinical symptoms, Tuberculin Skin Test (TST), chest X-rays, and newer molecular tests like GeneXpert MTB/RIF.
- Diagnosis also involves identifying and screening children who are in close contact with TB patients.

2. Pediatric Drug Formulations:

- Provision of child-friendly TB medications, including fixed-dose combinations (FDCs) that are easier for children to take.
- Dosages are carefully calculated based on the child's age and weight to ensure efficacy and reduce side effects.

3. Treatment Regimens:

- Standardized treatment regimens for both drug-sensitive and drug-resistant TB.
- The treatment for drug-sensitive TB typically involves an initial intensive phase followed by a continuation phase.
- Management of drug-resistant TB requires longer and more complex treatment regimens with second-line drugs.

4. **Directly Observed Treatment (DOT):**

- Implementation of the DOT strategy to ensure adherence to the treatment regimen.
- Involves a healthcare worker or trained volunteer observing the child taking their TB medications.

5. **Nutritional Support and Counselling:**

- Recognition of the role of nutrition in TB management.
- Nutritional assessment and support are provided, along with Counselling for the child and family.

6. **Contact Tracing and Preventive Therapy:**

- Systematic evaluation of household and close contacts of TB patients, particularly children under 5 years of age.
- Administration of preventive therapy to children who are household contacts of pulmonary TB patients and are at high risk.

7. **Monitoring and Follow-Up:**

- Regular monitoring for treatment response, side effects, and adherence.
- Follow-up visits are scheduled at regular intervals during and after the completion of treatment.

8. **Training and Sensitization of Healthcare Providers:**

- Training programs for healthcare providers to enhance their skills in diagnosing and managing pediatric TB.
- Sensitization about the importance of child-specific approaches in TB care.

9. **Community Awareness and Stigma Reduction:**

- Efforts to increase community awareness about TB, its symptoms, and the importance of early treatment.
- Addressing stigma associated with TB to encourage families to seek timely medical help.

10. **Integration with Other Child Health Programs:**

- Integration of TB care with other child health services like immunization, HIV programs, and malnutrition interventions.

11. **Recording and Reporting:**

- Systematic recording and reporting of all pediatric TB cases as part of the national TB surveillance system.

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10. a) **What is child labour and discuss the measures to control it in the society?**

- ❖ Child labor refers to the exploitation of children through any form of work that deprives them of their childhood, their potential, and their dignity, and that is harmful to physical and mental development
- ❖ It involves either being under the legal age for work or working under conditions harmful to the child
- ❖ In the context of Indian society, child labor is a complex issue influenced by economic, social, and cultural factors.
- ❖ Controlling child labor in Indian society requires a multifaceted approach that addresses the root causes, including poverty, lack of education, and social norms
- ❖ It involves a combination of legal action, educational initiatives, economic support, and societal change
- ❖ The active participation of government bodies, NGOs, communities, and international organizations is crucial in the fight against child labor.

Measures to Control Child Labor in Indian Society:

1. **Strengthening Legal Framework:**

- Enforcing existing laws like the Child Labour (Prohibition and Regulation) Act, which prohibits the employment of children below the age of 14 in hazardous occupations and processes.
- Regular updating and implementation of laws to cover various forms of child labor and exploitation.

2. **Education Policies:**

- Implementing the Right to Education Act, which mandates free and compulsory education for all children aged 6 to 14 years.

- Enhancing the quality of education and making schools accessible and child-friendly to encourage attendance and reduce dropout rates.

3. **Economic Measures:**

- Addressing poverty, a key driver of child labor, through social welfare programs, financial assistance, and livelihood opportunities for families.
- Promoting sustainable economic development to improve living standards and reduce reliance on child labor.

4. **Awareness and Social Campaigns:**

- Conducting awareness campaigns to educate the public about the negative impacts of child labor.
- Encouraging community participation in monitoring and reporting instances of child labor.

5. **Rehabilitation and Support Programs:**

- Providing rehabilitation services for children rescued from labor, including education, vocational training, and psychological support.
- Offering support programs for families of child laborers to prevent recurrence.

6. **Corporate Responsibility and Supply Chain Monitoring:**

- Encouraging businesses to adopt ethical practices that do not involve child labor.
- Monitoring supply chains, especially in industries prone to child labor, such as textiles, fireworks, and carpet making.

7. **Strengthening Labor Inspection and Law Enforcement:**

- Enhancing the capacity and resources of labor inspectors to enforce child labor laws effectively.
- Ensuring swift and strict legal action against those violating child labor laws.

8. **Partnerships and Collaborations:**

- Collaborating with NGOs, international organizations, and civil society groups to combat child labor.
- Engaging in partnerships to share resources, expertise, and best practices.

9. Empowering Women and Marginalized Communities:

- Focusing on empowering women through education and employment, as this has a direct impact on reducing child labor.
- Targeting assistance to marginalized communities where child labor is more prevalent.

10. Incentivizing Education:

- Providing incentives such as scholarships, mid-day meals, and free textbooks to encourage school attendance over work.

11. Community-Based Approaches:

- Involving local communities in identifying and addressing child labor issues.
- Establishing community vigilance groups to keep a check on child labor practices.

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10. b) How will you handle the Cyber bullying in children?

- ❖ Handling cyberbullying in children is a multifaceted challenge that requires a proactive and comprehensive approach
- ❖ It involves education, awareness, support, and appropriate intervention strategies
- ❖ Handling cyberbullying in children requires a balanced approach that combines education, support, vigilance, and appropriate intervention
- ❖ It's important to empower children with the knowledge and skills to protect themselves online, provide them with support and resources to deal with cyberbullying, and ensure there are effective mechanisms in place for reporting and addressing such incidents
- ❖ Collaboration among parents, schools, and the wider community is essential in creating a safe online environment for children.

Here are steps to effectively handle cyberbullying in children:

1. Education and Awareness:

- Educate children about what cyberbullying is and its harmful effects.

- Teach them about responsible online behavior and digital citizenship.
- Inform them about the importance of privacy settings and the risks of sharing personal information online.

2. **Open Communication:**

- Encourage open and honest communication between children and adults.
- Create a safe and supportive environment where children feel comfortable discussing their online experiences and reporting any instances of cyberbullying.

3. **Recognizing the Signs:**

- Train parents, teachers, and caregivers to recognize signs of cyberbullying, such as changes in the child's behavior, reluctance to use electronic devices, or signs of emotional distress.

4. **Immediate Response:**

- If a child is being cyberbullied, take immediate action. This may include saving the evidence, blocking the bully, and reporting the behavior to the relevant platforms.
- In serious cases, it may be necessary to involve school authorities or law enforcement.

5. **Support and Counselling:**

- Provide emotional support and Counselling to the child victim. Addressing the psychological impact is crucial.
- Engage professional help if necessary, especially if the child shows signs of anxiety, depression, or other emotional issues.

6. **Involving Schools:**

- Collaborate with schools to address cyberbullying. Many schools have policies and programs in place.
- Schools can conduct workshops, assemblies, and classroom sessions on cyberbullying and internet safety.

7. **Parental Involvement:**

- Encourage parents to be involved in their children's online activities.

- Teach parents how to use parental controls and monitor their child's online presence in a non-invasive way.

8. **Policy and Legal Awareness:**

- Make children and parents aware of the legal implications of cyberbullying.
- Inform them about the policies and laws that protect against online harassment and abuse.

9. **Peer Support and Leadership:**

- Encourage peer support programs where children can support each other.
- Develop peer leadership programs that empower students to take a stand against cyberbullying.

10. **Promoting Positive Online Communities:**

- Encourage children to be part of positive online communities and engage in constructive online activities.
- Teach them to stand up against cyberbullying and support peers who may be victims.

11. **Regular Review of Online Habits:**

- Regularly review and discuss online habits with children to ensure they are engaging in safe and respectful online behavior.

12. **Building Resilience:**

- Work on building the child's resilience and self-esteem to help them cope with and respond to cyberbullying.

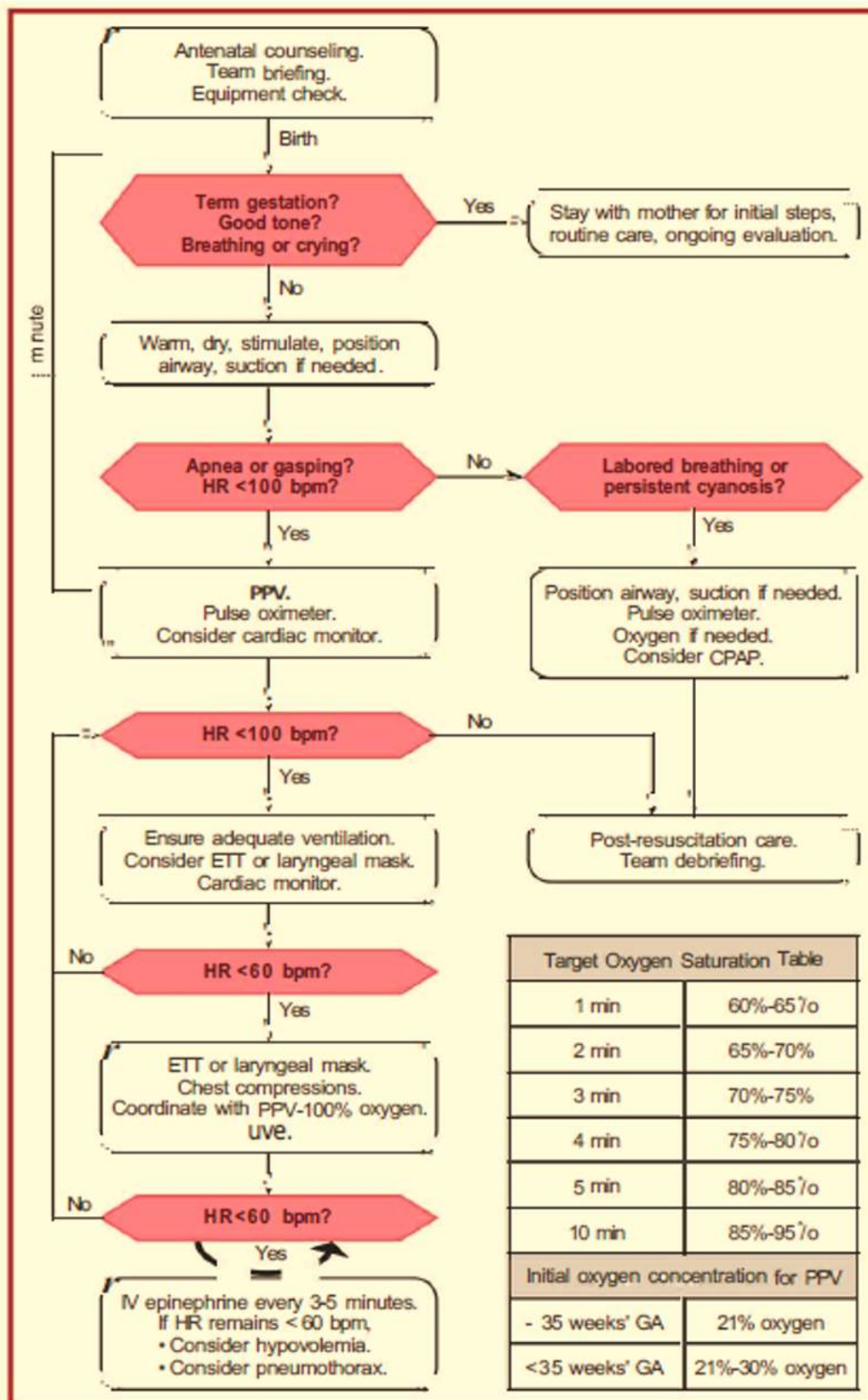
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Paper 2

1. a) Algorithm of neonatal resuscitation programme (NRP)



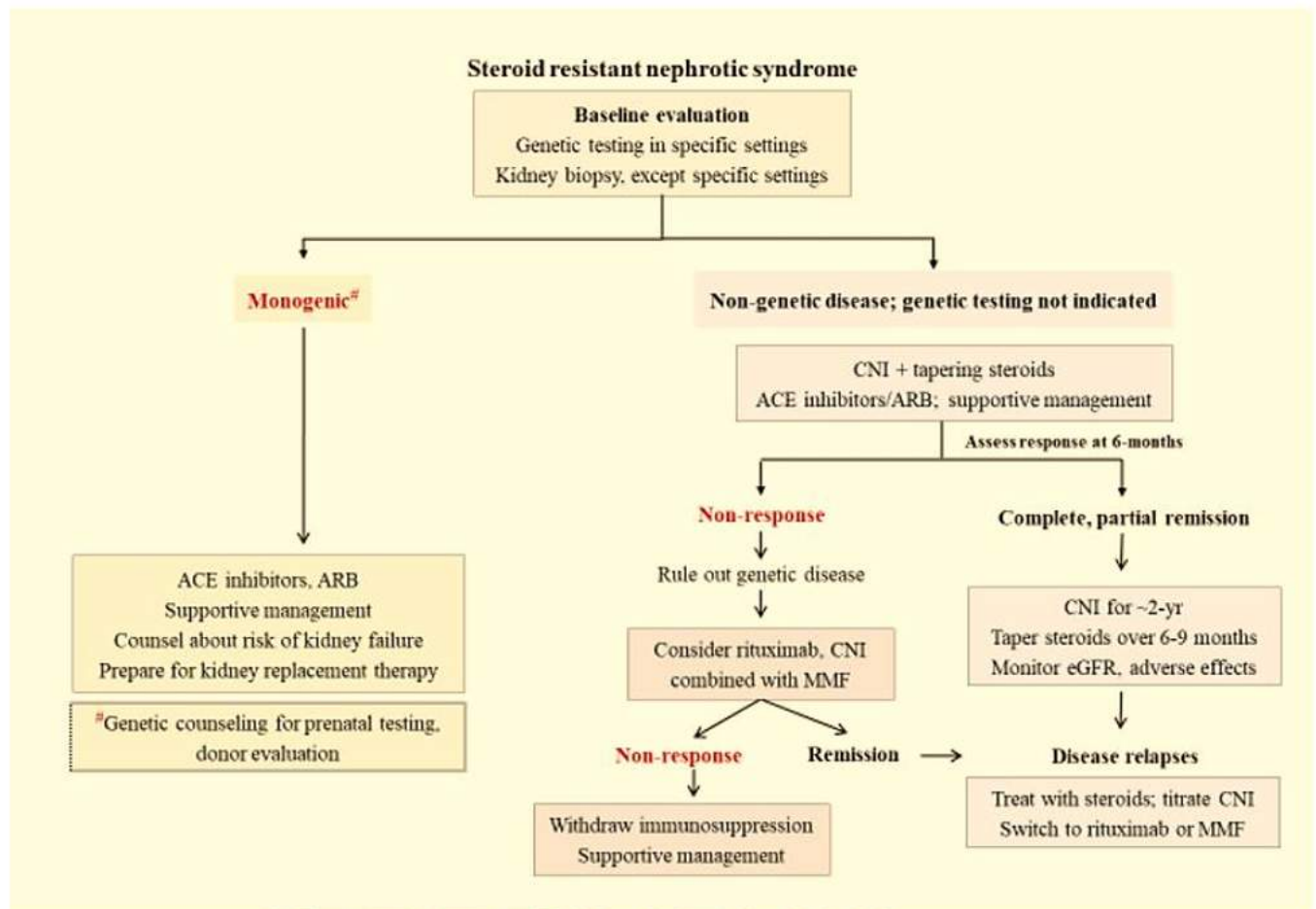
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1. b) Steroid resistant nephrotic syndrome.

- **Definition:** Steroid-Resistant Nephrotic Syndrome (SRNS) is characterized by the absence of remission despite four weeks of daily prednisolone therapy.
- **Initial Approach:**
 - Begin with genetic testing in specific cases and a kidney biopsy unless certain conditions are met.
 - For monogenic SRNS, consider genetic Counselling, ACE inhibitors, ARBs, and supportive management.
 - In non-genetic SRNS where genetic testing is not indicated, commence with Calcineurin Inhibitors (CNI) and tapering steroids, along with ACE inhibitors/ARBs and supportive management.
- **Assessment of Therapy:**
 - Evaluate response at 6 months.
 - If there's no response, rule out genetic disease, and consider adding rituximab, CNI combined with MMF (mycophenolate mofetil).
 - In cases of remission, continue CNI for approximately 2 years, taper steroids over 6-9 months, and monitor eGFR and for adverse effects.
 - In the case of relapse, treat with steroids, titrate CNI, or switch to rituximab or MMF.
- **Medication Dosing and Monitoring:**
 - **Tacrolimus:** 0.1-0.2 mg/kg/day in 2 divided doses; max initial dose 4 mg/day. Monitor blood levels.
 - **Cyclosporine:** 3-5 mg/kg/day in 2 divided doses; max initial dose 200 mg/day. Monitor blood levels.
 - **Prednisolone on alternate days:** 1.5 mg/kg for 4 weeks, then taper to 0.3-0.5 mg/kg for ~6-9 months.
 - **Cyclophosphamide:** 500-750 mg/m² IV every month for 6 months.
 - **Rituximab:** 375 mg/m² every 2 weeks for 2-4 doses.
 - **Mycophenolate mofetil:** 600-1200 mg/m² in divided doses.

- **Adverse Effects and Their Monitoring:**
 - Tacrolimus and Cyclosporine can cause nephrotoxicity and other systemic effects, requiring regular monitoring of renal function, blood pressure, and other parameters.
 - Prednisolone can cause weight gain, hypertension, and glucose intolerance, among other side effects.
 - Cyclophosphamide's adverse effects include leukopenia and hemorrhagic cystitis.
 - Rituximab has risks such as infusion reactions and infections.
 - Mycophenolate mofetil may lead to leukopenia, liver dysfunction, and gastrointestinal issues.
- **Monitoring Regimens:**
 - Screen for cosmetic side effects, tremors, diarrhea, and hypertension.
 - Regularly assess blood glucose, liver function tests, and blood counts.
 - Evaluate for opportunistic infections and check immunoglobulin levels with drugs like rituximab.
- **Further Considerations:**
 - In case of non-response to initial treatments, withdrawal of immunosuppression and supportive management is advised.
 - Continuous re-evaluation of therapy effectiveness and side effects is critical for managing SRNS effectively.
 - Disease relapse requires a return to aggressive treatment or an alteration in the therapeutic regimen.

The management of SRNS must be personalized, taking into consideration the patient's response to treatment, the side effects experienced, and any underlying genetic conditions.



2. a) Bone age assessment and physical growth in children.

Bone age assessment is a crucial tool in pediatric and adolescent medicine. It serves multiple purposes from diagnosis to treatment planning and monitoring, making it indispensable in managing growth-related conditions.

- **Available Methods for Determination of Bone Age**
 - **Radiographic Assessment (most commonly employed in daily practice)**
 - Greulich and Pyle Method: Involves comparing a single anteroposterior (AP) radiograph of the left hand and wrist to a standard atlas.
 - Tanner-Whitehouse Method: Considers multiple bones in the hand and wrist, assigning them maturity scores that are summed up.

- Birth- knee joint- lower end of femur;
- Infancy – shoulder joint
- 2-14 yrs – wrist joint
- 12-14 yrs – elbow,
- hip
- 15-18 yrs – wrist, Elbow and shoulder, hip
- > 17yrs – jaw for 3rd molar

Order of appearance of ossification centres – CARPAL BONES (CHTL – STTP)

Capitate	2 mon
Hamate	3 mon
Triquetrum	3 yrs
Lunate	4yrs
Scaphoid	5 yrs
Trapezium	5 yrs
Trapezoid	5 yrs
Pisiform	12 yrs

- **Automated Methods**

- BoneXpert: A software that automatically analyzes hand radiographs.
- Digital Atlas: Computer-based programs that compare patient radiographs to a digital atlas.

- **Ultrasound Imaging**

- Measures cortical thickness and other parameters in long bones.

- **Magnetic Resonance Imaging (MRI)**

- Used less frequently due to cost but offers detailed images without radiation exposure.

- **Dual-Energy X-ray Absorptiometry (DEXA)**

- Measures bone mineral density, but less commonly used for bone age assessment.

- **Clinical Examination**

- Assessment of secondary sexual characteristics, though less accurate.

- **Indications for Determination of Bone Age**

- Short Stature or Tall Stature: To evaluate growth potential.
- Pubertal Disorders: To assess the timing and progression of puberty.
- Endocrine Disorders: Conditions like hypothyroidism or growth hormone deficiency.
- Chronic Illness: In conditions like renal failure or malnutrition.

- Constitutional Growth Delay: To differentiate from pathological causes.
- Skeletal Dysplasias: To assess the severity and prognosis.
- Forensic Investigations: To estimate age in the absence of birth records.
- **Bone Age Assessment and Its Usefulness**
 - **Growth Prediction:** Helps in estimating adult height.
 - **Treatment Planning:** Guides therapeutic interventions like growth hormone therapy.
 - **Monitoring Therapy:** Evaluates the effectiveness of treatment over time.
 - **Diagnosis:** Assists in diagnosing various medical conditions affecting growth.
 - **Psychosocial Adjustment:** Helps in counselling children and families about growth expectations.
 - **Legal and Ethical Applications:** Useful in age determination in legal scenarios.

Physical growth in children:

It is a complex process that involves changes in body size, shape, and composition over time.

It is one of the most visible aspects of child development and is influenced by a combination of genetic, nutritional, environmental, and hormonal factors.

1. **Growth Phases:**

- **Infancy (0-2 years):** Rapid growth, with infants typically tripling their birth weight by the end of the first year.

Physical Growth in infancy- rapid phase

- **Birth-3 Months:** Infants typically double their birth weight by 5 months. Initial weight loss in the first week is regained by the second week.
- **4-6 Months:** Rapid weight gain continues, and birth weight is usually doubled by this stage.
- **7-9 Months:** Slower weight gain but increased length; birth weight is tripled by the end of the first year.

- **10-12 Months:** Growth in length is about 50% more than the length at birth.

Age	Weight gain	Length gain
0-3 months	30 gm/day	3.5 cms/month
3-6 months	20gm/day	2 cms/month
6-9 months	15gm/day	1.5 cms/month
9-12 months	12gm/day	1.2 cms/month
1-3 yrs	8gm/day	

Height/Length gain	
0-6 months	16 cms
6-12 months	9 cms
1-2 yrs	12.5cms
2-5 yrs	12 cms/yr
5-10yrs	5-6 cms/yr

Weight	Age	Average values
x	Birth	3kg
2x	5 months	6 kg
3x	1 year	9 kg
4x	2-2.5 years	12 kg
5x	3 years	15 kg

Length/Height	Age	Average values
y	Birth	50 cms
1.5 y	1 year	75 cms
1.75 y	2 years	87.5 cms
2 y	4 years	100 cms

Age	OFC	Gain	Average values
Birth			35 cms
1-2weeks	Shrinks and returns		35 -36 cms
0-3 months	0.5 cms/week = 2cms/month	6 cms	41 cm
3 -6 months	0.25 cms/week = 1 cms/month	3 cms	44 cms
6-12 months	0.5 cms/month	3 cms	47 cms
		Total =12 cms in 1 yr	

- **Early Childhood (2-6 years):** Slower, steady growth in height and weight.

- **Middle Childhood (6-11 years):** Continued steady growth; preparation for the growth spurt of puberty.
- **Adolescence (12-18 years):** Pubertal growth spurt; significant changes in height, weight, and body composition.

2. **Growth Measurements:**

- **Height/Length:** Measured using a stadiometer or length board.
- **Weight:** Measured using a standard scale.
- **Head Circumference:** Important in infants and toddlers to monitor brain growth.
- **Body Mass Index (BMI):** Used in older children to assess weight relative to height.

3. **Growth Charts and Percentiles:**

- Growth charts (such as WHO or CDC charts) are used to track a child's growth over time.
- Growth is plotted on percentile curves to compare with peers and assess patterns of growth.

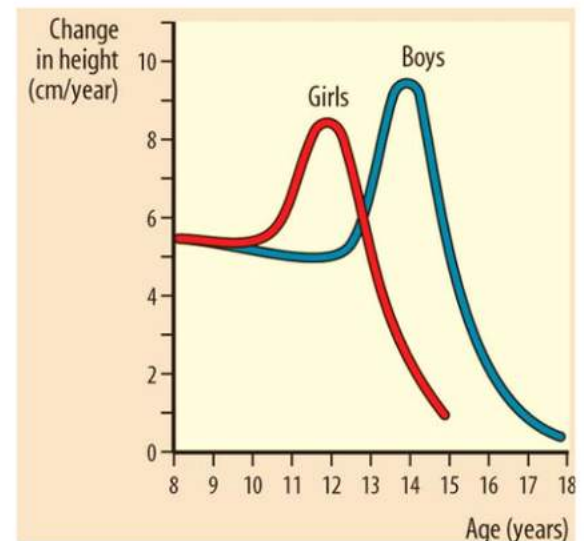
4. **Factors Influencing Growth:**

- **Genetics:** Major determinant of a child's growth potential.
- **Nutrition:** Adequate nutrition is crucial for normal growth; malnutrition can lead to growth retardation.
- **Hormones:** Growth hormone, thyroid hormones, and sex hormones play key roles in growth.

- **Health Status:** Chronic illnesses or conditions can affect growth.
- **Socioeconomic Factors:** Access to healthcare, nutrition, and living conditions impact growth.

5. **Pubertal Growth Spurt:**

- Occurs during adolescence; characterized by rapid increase in height and weight.
- Girls typically enter puberty earlier than boys and complete their growth earlier.



6. **Monitoring Growth:**

- Regular monitoring is essential to identify normal growth patterns and detect any deviations.
- Pediatricians use growth charts and physical examinations to assess growth.

7. **Abnormal Growth Patterns:**

- **Growth Failure:** Falling significantly below the normal growth range for age and sex.
- **Precocious Puberty:** Early onset of puberty.
- **Delayed Growth:** Slower than normal growth rate.

8. **Interventions for Abnormal Growth:**

- Addressing underlying causes (e.g., nutritional deficiencies, hormonal imbalances).
- In cases of growth hormone deficiency, growth hormone therapy may be considered.

9. **Importance of Healthy Lifestyle:**

- Balanced diet, regular physical activity, and adequate sleep are important for normal growth.
- Emotional well-being also plays a role in a child's physical development.

10. **Cultural and Ethnic Variations:**

- Growth patterns can vary by ethnicity and geographic location.

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2. b) Point of Care (POC) tests for the diagnosis of Urinary Tract Infections (UTIs)

- ❖ They are rapid diagnostic tools used in clinical settings to quickly detect the presence of a UTI
- ❖ These tests are particularly useful because they provide immediate results, allowing for prompt treatment decisions
- ❖ POC tests for UTIs are valuable tools in the rapid diagnosis and management of urinary tract infections
- ❖ They offer the advantage of speed and convenience, which is particularly important in acute care settings
- ❖ However, they should be used in conjunction with clinical judgment and, in some cases, followed up with more definitive laboratory testing.

Here are some commonly used POC tests for UTIs:

1. **Urinalysis:**

- **Dipstick Test:** A simple, rapid test using a chemically treated strip that is dipped into a urine sample. It tests for:
 - **Nitrites:** Indicates the presence of nitrate-reducing bacteria, commonly associated with UTIs.
 - **Leukocyte Esterase:** Suggests the presence of white blood cells in the urine, indicative of an infection.
 - **Blood:** Can be a sign of UTI or other urinary tract disorders.
 - **Protein, pH, and Specific Gravity:** Additional parameters that can support the diagnosis.

2. **Microscopic Urinalysis:**

- Examining a urine sample under a microscope to detect:
 - **White Blood Cells (Pyuria):** Suggests inflammation, often due to infection.
 - **Red Blood Cells:** Can be present in UTIs or other renal conditions.

- **Bacteria and Yeasts:** Direct visualization of pathogens can support the diagnosis.

3. **Rapid Urine Culture:**

- Some newer POC devices can provide rapid urine culture results, though these are less common in routine clinical practice compared to standard laboratory cultures.
- These tests can identify bacterial growth more quickly than traditional culture methods.

4. **Biosensors and Automated Systems:**

- Advanced POC testing systems use biosensor technology to detect bacteria and leukocytes in urine.
- These systems can provide results in a matter of minutes and are becoming more prevalent in some healthcare settings.

5. **Nucleic Acid Amplification Tests (NAATs):**

- Rapid tests that can detect bacterial DNA or RNA in urine samples.
- Highly sensitive and specific, but more expensive and typically used in complex cases or where rapid identification of specific pathogens is crucial.

6. **Portable Scanning Devices:**

- Handheld or portable devices that can analyze urine samples and provide immediate results.
- These devices are increasingly being developed for POC use, utilizing various technologies like digital imaging and machine learning algorithms.

Advantages of POC Tests for UTIs:

- **Rapid Results:** Provide immediate information, facilitating prompt treatment.
- **Convenience:** Can be performed in various settings, including clinics, emergency departments, and potentially at home.
- **Reduced Need for Laboratory Testing:** Can decrease the reliance on time-consuming laboratory cultures for initial diagnosis.

Limitations:

- **Sensitivity and Specificity:** While convenient, POC tests may not be as sensitive or specific as traditional urine cultures.
- **Interpretation:** Results may require interpretation by trained personnel.
- **Cost and Availability:** Some advanced POC tests may be more expensive or not readily available in all healthcare settings.

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3.a) Management of transfusion-dependent thalassemia (TDT)

Thalassemia major, a severe form of thalassemia, often requires regular blood transfusions to manage anemia and maintain adequate hemoglobin levels.

1. **Regular Blood Transfusions:**

- Aim to maintain pre-transfusion hemoglobin levels above 9-10.5 g/dL.
- Transfusion intervals typically range from every 2 to 5 weeks, depending on individual needs.
- Use of leukoreduced, matched (for Rh and Kell antigens) packed red blood cells is recommended to reduce alloimmunization and transfusion reactions with CMV filters.

2. **Iron Overload Monitoring and Management:**

- Chronic transfusions lead to iron overload, a major complication in TDT.
- Regular monitoring of serum ferritin and liver iron concentration (LIC) using MRI T2*.
- Initiation of iron chelation therapy when serum ferritin consistently exceeds 1000 ng/mL or LIC is high.

3. **Iron Chelation Therapy:**

- Essential to prevent iron-induced organ damage.
- Common chelators include Deferoxamine (administered subcutaneously or intravenously), Deferasirox (oral), and Deferiprone (oral).
- The choice and combination of chelators depend on the degree of iron overload, patient compliance, and side-effect profiles.

Iron Chelation Therapy

- **Rationale:** Chronic blood transfusions lead to iron overload, which can cause multi-organ failure. Iron chelation is essential for mitigating these risks.
- **Types of Chelators:**
 - **Deferoxamine (Desferal):**
 - **Administration:** Subcutaneous infusion over 8-12 hours, 5-7 days a week.
 - **Dosage:** 20-50 mg/kg/day.
 - **Monitoring:** Audiometry and ophthalmology exams due to risk of hearing and vision changes.
 - **Deferasirox (Exjade, Jadenu):**
 - **Administration:** Oral, once daily.
 - **Dosage:** 20-40 mg/kg/day.
 - **Monitoring:** Regular liver and kidney function tests; gastrointestinal assessments.
 - **Deferiprone (Ferriprox):**
 - **Administration:** Oral, three times daily.
 - **Dosage:** 75-100 mg/kg/day.
 - **Monitoring:** Weekly complete blood counts due to risk of agranulocytosis.
- **Combination Therapy:**
 - **Deferoxamine and Deferiprone:** Synergistic effect; used in patients with cardiac iron overload.
- **Monitoring and Assessment:**
 - **Serum Ferritin:** Monthly; target levels below 1000 µg/L.
 - **Liver Iron Concentration:** Via MRI or liver biopsy.
 - **Cardiac MRI T2*:** To assess cardiac iron overload.
- **Adverse Effects:**
 - **Deferoxamine:** Ototoxicity, ocular toxicity, and allergic reactions.

- **Deferasirox:** Gastrointestinal issues, renal impairment, and hepatic dysfunction.
- **Deferiprone:** Neutropenia and agranulocytosis.

4. **Regular Monitoring for Complications:**

- Monitoring for complications related to iron overload, including cardiac, liver, and endocrine dysfunctions.
- Regular echocardiography and endocrine evaluations are recommended.

5. **Infection Prevention and Management:**

- Patients with TDT are at increased risk of infections, including transfusion-transmitted infections and those related to iron overload.
- Regular screening for transfusion-transmitted infections and appropriate vaccinations are important.

6. **Splenectomy Considerations:**

- Splenectomy may be considered in cases of hypersplenism (excessive destruction of blood cells by the spleen) or to reduce transfusion requirements.
- However, it increases the risk of infection and thrombosis, so prophylactic antibiotics and possibly anticoagulation may be required post-splenectomy.

7. **Bone Health Management:**

- Monitoring and managing bone health due to increased risk of osteoporosis and fractures.
- Includes adequate calcium and vitamin D intake, regular exercise, and bone density monitoring.

8. **Psychosocial Support:**

- Ongoing psychological support for patients and families to cope with the chronic nature of the disease and its treatment.

9. **Advanced Therapies:**

- Gene therapy is an emerging treatment modality and is under investigation.

- Hematopoietic stem cell transplantation (HSCT) may be considered for eligible patients, offering the potential for a cure.

Hematopoietic Stem Cell Transplantation (HSCT)

- **Rationale:** The only curative treatment for Thalassemia Major; replaces the defective hematopoietic stem cells with healthy ones from a donor.
- **Donor Selection:**
 - **HLA-Matched Sibling:** Ideal choice.
 - **Unrelated Donor:** Alternative; requires stringent HLA matching.
- **Conditioning Regimens:**
 - **Myeloablative:** Busulfan, cyclophosphamide, and anti-thymocyte globulin.
 - **Reduced-Intensity:** Fludarabine-based regimens; less toxic but higher risk of graft failure.
- **Transplantation Procedure:**
 - **Source:** Bone marrow or peripheral blood stem cells.
 - **Infusion:** Intravenous infusion of stem cells.
- **Post-Transplant Care:**
 - **Graft-Versus-Host Disease (GVHD) Prophylaxis:** Immunosuppressive medications like cyclosporine.
 - **Infection Control:** Antimicrobial prophylaxis and isolation.
- **Monitoring and Assessment:**
 - **Chimerism Studies:** To assess the engraftment status.
 - **Immunological Reconstitution:** Monitoring of immune cell counts.
- **Risks and Complications:**
 - **GVHD:** Acute or chronic; affects skin, liver, and gastrointestinal tract.
 - **Infections:** Due to immunosuppression.
 - **Graft Failure:** Rejection of the transplanted cells.

10. Lifestyle and Dietary Considerations:

- Encouraging a healthy lifestyle, including a balanced diet and avoidance of excess iron intake.

11. **Regular Follow-Up and Comprehensive Care:**

- Regular follow-up with a multidisciplinary team including hematologists, cardiologists, endocrinologists, and other specialists as needed.

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3. b) **Diagnosis of tuberculous meningitis**

• **Clinical Suspicion of TBM:**

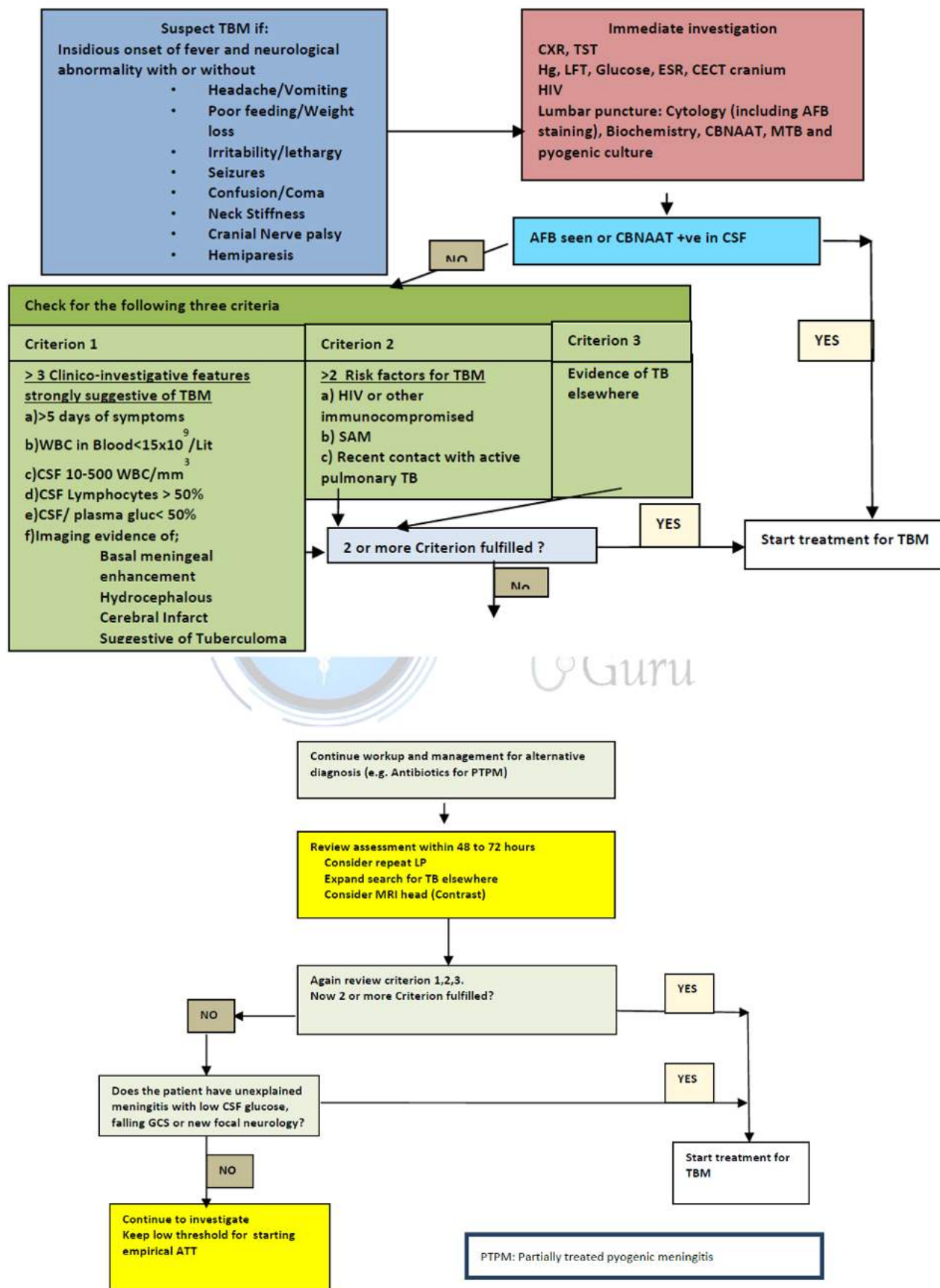
- Presentation: Suspect TBM in children with insidious onset of fever and neurological abnormality or other suggestive symptoms such as:
 - Headache/Vomiting
 - Poor Weight
 - Loss of consciousness
 - Irritability/lethargy
 - Seizures
 - Confusion/Coma
 - Neck Stiffness
 - Cranial nerve palsy
 - Hemiparesis

• **Immediate Investigations:**

- Undertake the following diagnostic measures:
 - Chest X-ray
 - Tuberculin Skin Test (TST)
 - CSF examination for Lymphocyte count, Glucose, Protein, and Chloride
 - Lumbar Puncture for AFB staining, culture, CBNAAT (Cartridge Based Nucleic Acid Amplification Test), and MTB PCR (Mycobacterium Tuberculosis Polymerase Chain Reaction)

- Brain imaging such as MRI or CT to detect hydrocephalus, basal meningeal enhancement, tuberculoma, or cerebral infarct
- **CSF Findings:** Check for the following three criteria to help establish the diagnosis:
 - **Criterion 1:**
 - Strongly suggestive clinical and investigative features:
 - 3 Clinical-unexplained fever >5 days of symptoms
 - Raised WBC in Blood >15000/ μ L
 - CSF lymphocyte count >50%
 - CSF protein >50mg%
 - CSF/plasma glucose <50%
 - Imaging-based evidence of basal meningeal enhancement
 - Evidence of hydrocephalus
 - Cerebral infarct
 - Presence of tuberculoma
 - **Criterion 2:**
 - Risk factors for TBM:
 - Contact with a known TB patient
 - Immunocompromised status
 - Recent contact with someone diagnosed with active pulmonary TB
 - **Criterion 3:**
 - Evidence of TB elsewhere in the body
- **Management Based on Criteria:**
 - If Acid-Fast Bacilli (AFB) is seen on CSF smear or CBNAAT is positive for MTB in the CSF, then start treatment for TBM immediately.
 - If two or more of the criteria are fulfilled, start treatment for TBM.

- If fewer than two criteria are met, continue the workup and manage for an alternative diagnosis (e.g., Antibiotics for Partially Treated Pyogenic Meningitis).
- **Follow-up Assessment:**
 - Re-evaluate the patient after 48 to 72 hours:
 - Consider repeating the lumbar puncture if required.
 - Expand the search for TB elsewhere in the body.
 - Consider the Mantoux Tuberculin Skin Test.
 - Review the earlier criteria (1,2,3) once again.
 - If now two or more criteria are met, proceed to initiate treatment for TBM.
- **Further Clinical Assessment:**
 - If the child has unexplained meningeal signs with low Glasgow Coma Scale scores (GCS) or new focal neurological signs, consider initiating treatment for TBM.
- **Subsequent Steps:**
 - If TB Meningitis is still not confirmed after the above steps, continue to investigate and keep the threshold low for starting empirical Anti-Tubercular Treatment (ATT).
- **Note:** The abbreviation 'PTPM' refers to Partially Treated Pyogenic Meningitis.



4. a) Overview of learning disability disorders.

- ❖ Learning disabilities are neurodevelopmental disorders that affect the brain's ability to receive, process, analyze, or store information
- ❖ These disabilities can cause a person to have trouble learning and using certain skills
- ❖ Each learning disability has its unique challenges and requires specific interventions and support
- ❖ Early identification and intervention, along with a supportive educational environment, are key to helping individuals with learning disabilities achieve their potential.

The table below summarizes various learning disability disorders, their characteristics, and typical interventions:

<i>Learning Disability</i>	Characteristics	Typical Interventions
<i>Dyslexia</i>	Difficulty in reading, writing, spelling, and speaking. Challenges in recognizing words, decoding letters to sound, and understanding written words.	Structured literacy programs focusing on phonics. Multisensory teaching approaches. Use of text-to-speech and speech-to-text software.
<i>Dyscalculia</i>	Difficulty in understanding numbers and mathematical concepts. Challenges in performing arithmetic operations and understanding time and money.	Use of visual and hands-on learning tools for math. Breaking down math problems into smaller, manageable steps. Teaching mathematical concepts using real-life examples.
<i>Dysgraphia</i>	Difficulty with handwriting, spelling, and organizing ideas. Poor handwriting, trouble holding a pencil correctly, and difficulty organizing thoughts on paper.	Occupational therapy for fine motor skills. Use of graphic organizers for writing. Permission to use computers for typing.
<i>Dyspraxia (Developmental Coordination Disorder)</i>	Difficulty with motor skills and coordination. Challenges with tasks requiring coordination, such as writing or riding a bike.	Physical therapy for motor skills. Occupational therapy for daily living skills. Adapted physical education programs.
<i>Auditory Processing Disorder (APD)</i>	Difficulty in processing auditory information.	Auditory training and therapy. Use of assistive listening devices.

	Challenges in understanding spoken language, especially in noisy environments.	Environmental modifications to reduce background noise.
Visual Processing Disorder	Difficulty in processing visual information. Challenges with understanding information seen, distinguishing shapes, or remembering visual details.	Visual therapy exercises. Use of visual organizers and aids. Adaptations in text size and color contrast for reading materials.
Nonverbal Learning Disability	Difficulty with nonverbal cues, such as facial expressions and body language. Challenges in spatial awareness, motor skills, and social skills.	Social skills training. Occupational and physical therapy for motor skills. Support in developing organizational skills.
Attention Deficit Hyperactivity Disorder (ADHD)	Difficulty in maintaining attention, hyperactivity, and impulsiveness. Challenges in staying focused, sitting still, and controlling behavior.	Behavioral therapy and interventions. Educational accommodations and support. Medication in some cases.

- **Individualized Education Program (IEP):** Most children with learning disabilities benefit from an IEP, which is a tailored educational plan developed to meet their specific needs.
- **Multidisciplinary Approach:** Involves educators, psychologists, speech and language therapists, occupational therapists, and other specialists.
- **Parental and Family Support:** Essential for the child's success, including advocacy and involvement in educational planning.

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4. b) Craniosynostosis

Definition of Craniosynostosis

- **Craniosynostosis:** A congenital condition in which one or more of the fibrous sutures in an infant's skull prematurely fuses, restricting skull growth and potentially leading to increased intracranial pressure and abnormal head shape.

Types of Craniosynostosis and Clinical Characteristics

Type of Craniosynostosis	Clinical Characteristics	Notes	Associated Syndromes
Sagittal	Elongated, narrow skull (scaphocephaly)	Most common type; affects the main suture on the top of the head	Often non-syndromic
Coronal	Flattening of the forehead on one or both sides (anterior plagiocephaly)	Second most common; affects the suture running from ear to ear	Apert, Crouzon, Pfeiffer
Metopic	Triangular forehead (trigonocephaly)	Affects the suture running from the nose to the top of the skull	Often non-syndromic
Lambdoid	Flattening at the back of the head (posterior plagiocephaly)	Least common; affects the suture at the back of the skull	Saethre-Chotzen, Carpenter

Syndromes Associated with Craniosynostosis

- Apert Syndrome
- Crouzon Syndrome
- Pfeiffer Syndrome
- Saethre-Chotzen Syndrome
- Carpenter Syndrome etc.

Syndrome	Genetic Cause	Clinical Features	Treatment Options
Saethre-Chotzen Syndrome	TWIST1 or FGFR2 genes	Craniofacial abnormalities; Limb malformations; Short stature; Intellectual disability; Hearing loss	Surgery for craniofacial abnormalities; Physical therapy for limb malformations

Antley-Bixler Syndrome	FGFR2 or FGFR3 genes	Craniofacial abnormalities; Joint contractures; Respiratory distress; Genital abnormalities	Surgery for craniofacial abnormalities; Physical therapy for joint contractures
Apert Syndrome	FGFR2 gene	Craniofacial abnormalities; Limb malformations; Intellectual disability; Hearing loss	Surgery for craniofacial and limb abnormalities; Physical therapy for developmental delays
Crouzon Syndrome	FGFR2 gene	Craniofacial abnormalities; Misshapen skull; Bulging eyes; Hearing loss; Dental problems	Surgery for craniofacial abnormalities; Hearing aids or cochlear implants for hearing loss
Pfeiffer Syndrome	FGFR1 or FGFR2 genes	Craniofacial abnormalities; Limb malformations; Hearing loss; Respiratory problems	Surgery for craniofacial and limb abnormalities; Respiratory support for breathing difficulties
Treacher Collins Syndrome	TCOF1 or POLR1C genes	Craniofacial abnormalities; Small jaw and cheekbones; Hearing loss; Dental problems	Surgery for craniofacial abnormalities; Hearing aids or cochlear implants for hearing loss
Stickler Syndrome	COL2A1, COL11A1, or COL11A2 genes	Craniofacial abnormalities; Cleft palate; Nearsightedness; Joint problems; Hearing loss	Surgery for craniofacial abnormalities; Glasses or contact lenses for vision problems; Physical therapy for joint problems

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5. a) Approach to a child with recurrent wheezing.

- ❖ The approach to a child with recurrent wheezing involves a comprehensive assessment to determine the underlying cause and appropriate management strategies.
- ❖ Recurrent wheezing is common in children and can be due to various reasons, including asthma, viral infections, anatomical anomalies, and other medical conditions.
- ❖ A systematic approach, including a detailed history, physical examination, and appropriate investigations, is essential for accurate diagnosis and management.

1. **Detailed Medical History:**

- Frequency, duration, and severity of wheezing episodes.
- Associated symptoms (e.g., cough, breathlessness, chest tightness).
- Triggers (e.g., exercise, allergens, cold air, infections).
- Family history of asthma or atopic diseases.
- Response to previous treatments.

2. **Physical Examination:**

- Assess respiratory rate, oxygen saturation, use of accessory muscles.
- Listen for wheezing, crackles, or decreased air entry.
- Look for signs of atopy (e.g., eczema, allergic rhinitis).
- Evaluate for growth and developmental delays.

3. **Diagnostic Investigations:**

- **Pulmonary Function Tests (PFTs):** Spirometry in children old enough to perform the test (usually >5 years old).
- **Chest X-Ray:** To rule out structural abnormalities and other lung conditions.
- **Allergy Testing:** Skin prick tests or serum IgE levels to identify potential allergens.
- **Blood Tests:** Complete blood count (CBC) to check for eosinophilia; other tests based on clinical suspicion.

4. **Identifying Underlying Causes:**

- Asthma: The most common cause of recurrent wheezing.
- Viral-induced wheeze: Common in preschool children.
- Anatomical anomalies: Such as vascular rings, tracheomalacia.
- Other causes: Cystic fibrosis, foreign body aspiration, immune deficiencies.

5. **Management Strategies:**

- **Asthma:** Follow asthma guidelines for management, including the use of inhaled corticosteroids, bronchodilators, and leukotriene receptor antagonists.
- **Viral-induced wheeze:** Symptomatic treatment; bronchodilators may be helpful in some cases.
- **Allergy Management:** Avoidance of identified allergens, use of antihistamines or nasal corticosteroids for allergic rhinitis.
- **Education and Monitoring:** Educate the family about the condition, trigger avoidance, and the correct use of inhalers. Provide an action plan for exacerbations.

6. **Regular Follow-Up:**

- Monitor response to treatment, growth, and development.
- Adjust treatment based on control of symptoms and side effects.

7. **Referral to Specialists:**

- Consider referral to a pediatric pulmonologist or allergist for complex cases or if the diagnosis is uncertain.
- Referral for further diagnostic evaluation in cases of suspected anatomical anomalies or other rare causes.

8. **Preventive Measures:**

- Vaccinations: Ensure up-to-date vaccinations, including the influenza vaccine.
- Exposure to tobacco smoke: Advise against smoking in the child's environment.

For addressing the differential diagnoses:

Condition	Key Features	Distinguishing Factors
Asthma	Episodic wheezing, cough, dyspnea. Triggered by allergens, exercise, cold air.	Family history of atopy. Good response to bronchodilators.
Viral Bronchiolitis	Common in infants and young children. Associated with viral upper respiratory infections.	Seasonal occurrence, often in winter. Rapid onset with respiratory distress.
Foreign Body Aspiration	Sudden onset of wheezing in a previously healthy child. May have a history of choking.	Unilateral wheezing or decreased breath sounds. No response to asthma medications.
Gastroesophageal Reflux Disease (GERD)	Wheezing, cough, especially postprandial or nocturnal. May have symptoms of acid reflux.	Symptoms improve with anti-reflux treatment. May coexist with asthma.
Tracheomalacia/Bronchomalacia	Congenital weakness of the trachea/bronchial walls. Wheezing, stridor, barking cough.	Symptoms present from early infancy. May improve with age.
Cystic Fibrosis	Persistent wheezing, cough with sputum. Failure to thrive, recurrent infections.	Positive family history. Positive sweat chloride test.
Vocal Cord Dysfunction	Paradoxical vocal cord movement. Stridor, throat tightness, voice changes.	Symptoms can mimic asthma but do not respond to bronchodilators. Diagnosed by laryngoscopy.
Allergic Rhinitis/Sinusitis	Nasal symptoms, postnasal drip, cough. Seasonal or perennial allergies.	Associated with other allergic symptoms. Response to antihistamines or nasal corticosteroids.
Cardiac Asthma	Wheezing due to heart failure. Associated with exercise intolerance, fatigue.	Cardiac history or findings on examination. Abnormal cardiac investigations.

Bronchopulmonary Dysplasia	In preterm infants with a history of prolonged mechanical ventilation. Chronic respiratory distress, wheezing.	History of prematurity and neonatal intensive care unit stay. Abnormal chest X-ray findings.
Immunodeficiency Disorders	Recurrent infections, wheezing, failure to thrive.	History of severe, recurrent, or unusual infections. Laboratory evidence of immunodeficiency.

Notes:

- **History and Physical Examination:** A thorough history and physical examination are crucial in differentiating these conditions.
- **Diagnostic Testing:** May include chest X-ray, pulmonary function tests, allergy testing, sweat chloride test, or bronchoscopy, depending on the suspected diagnosis.
- **Multidisciplinary Approach:** In complex cases, collaboration with specialists such as pulmonologists, allergists, gastroenterologists, or cardiologists may be necessary.

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5. b) Treatment of nutritional rickets

Age Group	Vitamin D Supplementation	Calcium Supplementation
Infants (0-12 months)	400-1,000 IU/day	200-260 mg/day
Children (1-18 years)	600-1,000 IU/day	700-1,300 mg/day
Adults (19-50 years)	600-4,000 IU/day	1,000 mg/day
Pregnant and Lactating Women	600-4,000 IU/day	1,000-1,300 mg/day

High-Dose Vitamin D Regimen for Severe Deficiency

- Stoss therapy: Single oral dose of 50,000 to 600,000 IU of vitamin D
- Or IM dose 300,000 to 600,000 IU single dose

- Or Oral 60,000 IU per dose weekly for 10 doses
- Followed by maintenance therapy: 400-1,000 IU/day

Calcium Supplementation in Severe Cases

- Calcium gluconate 10% solution: 0.11-0.22 mmol/kg IV over 10-30 minutes
- Followed by maintenance therapy: 1-2 g elemental calcium/day orally

Monitoring

- Serum calcium and phosphate levels should be monitored to avoid hypercalcemia or hyperphosphatemia.
- 25-hydroxyvitamin D levels should be checked to assess the efficacy of treatment.

Management Component	Description	Dosage/Duration
Vitamin D Supplementation	Essential for calcium absorption and bone mineralization	High-dose regimen often initiated, followed by maintenance doses
Calcium Supplementation	To correct hypocalcemia and support bone health	Dosage varies based on age and severity of deficiency
Phosphorus Supplementation	Rarely needed but can be used in cases of hypophosphatemia	Dosage varies; caution due to risk of hyperphosphatemia
Alkaline Phosphatase Monitoring	Marker of bone turnover; used to assess treatment efficacy	Regular monitoring until levels normalize
Parathyroid Hormone (PTH) Monitoring	Elevated in rickets; used to assess treatment efficacy	Regular monitoring until levels normalize
Pain Management	For symptomatic relief from bone pain	NSAIDs or other analgesics as needed
Physical Therapy	To improve muscle tone and correct deformities	As advised by a physiotherapist
Surgical Intervention	For severe deformities unresponsive to medical treatment	Procedures like osteotomy may be considered

Monitoring and Follow-up	Regular assessments to evaluate treatment efficacy and adjust management	Includes clinical, radiological, and biochemical evaluations
Dietary Counselling	To ensure adequate intake of vitamin D and calcium	Individualized based on dietary habits and nutritional needs
Patient Education	To ensure compliance and prevent recurrence	Ongoing education on the importance of vitamin D and calcium

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6. a) Staging & management hypoxic ischemic encephalopathy (HIE)

The Sarnat and Sarnat staging of Hypoxic-Ischemic Encephalopathy (HIE) in neonates, developed by Harold Sarnat and Selma Sarnat in 1976, is a clinical grading system used to classify the severity of HIE based on neurological symptoms.

This system is widely used due to its simplicity and effectiveness in assessing the extent of brain injury for more than 36 weeks. The staging is as follows:

Characteristic	Stage I (Mild HIE)	Stage II (Moderate HIE)	Stage III (Severe HIE)
Duration	< 24 hours	24 hours to several days	May last weeks
Consciousness	Hyperalert	Lethargic or obtunded	Stupor or coma
Muscle Tone	Normal to slightly increased	Hypotonia (decreased muscle tone)	Flaccid
Stretch Reflexes	Overactive	Decreased	Absent
Autonomic Function	Mydriasis (dilated pupils); Tachycardia	Bradycardia; Miosis (constricted pupils)	Variable; often dysautonomia (unstable temperature, blood pressure, heart rate)
Seizures	Absent	Common, often focal	Frequent, often multifocal or status epilepticus

Prognosis	Generally favorable; complete recovery expected	Variable; risk of long-term neurological deficits	Poor; high risk of mortality and severe disability
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Levene classification which is applicable for both term and preterm babies

Characteristic	Mild HIE	Moderate HIE	Severe HIE
Consciousness	Irritable	Lethargy	Comatose
Tone	Hypotonia	Marked hypotonia	Severe hypotonia
Seizures	No	Yes	Prolonged
Sucking/Respiration	Poor suck	Unable to suck	Unable to sustain spontaneous respiration

Management of HIE:

Initial Stabilization and Supportive Care:

- **Resuscitation:** Immediate neonatal resuscitation to establish breathing and circulation is crucial. This may involve clearing the airways, providing supplemental oxygen, and, if necessary, intubation and mechanical ventilation.
- **Hemodynamic Support:** Maintenance of adequate perfusion and blood pressure is essential to ensure oxygen and nutrient delivery to the brain and other organs.
- **Thermoregulation:** Maintaining normal body temperature is important, as hyperthermia can exacerbate brain injury.
- **Monitoring:** Continuous monitoring of vital signs, blood gases, glucose, and electrolyte levels is required to promptly identify and correct any abnormalities.

Therapeutic Hypothermia:

- **Indication:** Indicated for infants with moderate to severe HIE if initiated within 6 hours of birth.
- **Method:** The infant's core body temperature is reduced to 33-34°C for 72 hours using a cooling blanket – whole body cooling.

Who is eligible for hypothermia?

To undergo therapeutic hypothermia, a neonate should be ≥ 36 weeks and ≥ 1800 grams and should satisfy point **a & b** (stated below)

(a). At least one of the Following:

- Apgar score of ≤ 5 at 10 min after birth
- Continued need for Resuscitation (IPPV) at 10 min after birth
- Acidosis within 60 min of birth (Defined as any occurrence of umbilical cord or Arterial or Capillary pH < 7.00)
- Base deficit ≥ 16 mmol/L in umbilical cord or any blood sample within 1 hour of birth (arterial, venous or capillary)

Infants that meet criteria (a) should be assessed for whether they meet the neurological abnormality criteria (b)

(b) Seizures (or) moderate to severe encephalopathy

Moderate to severe encephalopathy consists of (all are required to be present):

- Altered state of consciousness (reduced or absent response to stimulation)
- Abnormal tone (focal or general hypotonia, or flaccid)
- Abnormal primitive reflexes (weak or absent suck or Moro response)

Note:

- Seizures detected by EEG or aEEG without a clinical component (subclinical seizures) would also be considered as seizures
- In babies who satisfy criteria (a) & (b), start therapeutic hypothermia at the earliest, but not later than 6 hours
- Examine and record a baseline (pre-TH) encephalopathy severity score (Thompson's score – Given at Section G in this chapter)

Who should not undergo Therapeutic Hypothermia:

- Gestational age < 36 weeks (confirmed by reliable LMP or early antenatal ultrasound or New Ballard scoring system – in this order of importance) (currently this is getting flexible with upcoming trials)

- Birth weight <1800 grams
 - Postnatal age greater than 6 hours
 - Presence of known chromosomal anomalies
 - Presence of major congenital malformations
 - Active bleeding (Bleeding from > 2 sites)
 - Grade 3 and above Intracranial hemorrhage (Therapeutic hypothermia not to be delayed if USG has not been done, but USG head to be done within 24 hours of birth and a decision regarding discontinuation of hypothermia has to be taken in Gr III IVH / Intra-Parenchymal Bleed)
 - Moribund neonate
- **Mechanism:** Hypothermia is believed to reduce metabolic demand, inhibit harmful biochemical cascades, and reduce cerebral edema.
 - **Monitoring During Hypothermia:** Continuous monitoring of core temperature, heart rate, blood pressure, oxygen saturation, and blood glucose levels is essential. Electroencephalography (EEG) may be used to monitor for seizures.
 - **Rewarming:** After 72 hours, the infant is slowly rewarmed to a normal body temperature at a controlled rate to prevent rebound cerebral edema or other complications.

Seizure Management:

- **Detection:** Seizures in neonates may be subtle; hence, continuous EEG monitoring is often used in infants with HIE.
- **Treatment:** Anticonvulsants such as phenobarbital or levetiracetam are used to control seizures. The choice of medication is based on seizure type, efficacy, and side-effect profile. Topiramate is promising due to its neuroprotective effects

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6.b) Approach to a newborn with respiratory distress

Respiratory distress in newborns can be due to a variety of causes, and prompt identification and management are crucial. Here's a structured approach:

1. **Initial Assessment:**

- **Airway:** Ensure the airway is clear. Look for obstructions or anomalies.
- **Breathing:** Assess respiratory rate, effort, and pattern. Note any grunting, flaring, or retractions.
- **Circulation:** Check heart rate, perfusion, and color. Look for cyanosis or pallor.
- **Disability:** Brief neurological assessment, including tone and activity level.

2. **History Taking:**

- **Prenatal History:** Maternal health, medications, infections.
- **Birth History:** Gestational age, type of delivery, birth weight, Apgar scores.
- **Postnatal History:** Onset and progression of respiratory distress, feeding difficulties, temperature instability.

3. **Physical Examination:**

- **General Appearance:** Activity level, posture, and tone.
- **Respiratory Examination:** Auscultation for breath sounds, wheezing, crackles.
- **Cardiovascular Examination:** Heart sounds, murmurs.
- **Abdominal Examination:** Distension, hepatomegaly.
- **Neurological Examination:** Reflexes, muscle tone.

4. **Differential Diagnosis:** (Details in table below)

- **Transient Tachypnea of the Newborn (TTN):** Common in term or near-term infants, often related to delayed clearance of lung fluid.
- **Respiratory Distress Syndrome (RDS):** Typically in preterm infants, due to surfactant deficiency.
- **Meconium Aspiration Syndrome:** In term or post-term infants with a history of meconium-stained amniotic fluid.
- **Pneumonia:** Considered in cases of maternal infection, prolonged rupture of membranes.
- **Congenital Anomalies:** Diaphragmatic hernia, airway malformations.

- **Pulmonary Hypertension:** May be primary or secondary to other conditions.

5. **Diagnostic Investigations:**

- **Chest X-Ray:** To evaluate lung fields, heart size, and position.
- **Blood Gases:** To assess oxygenation and ventilation status.
- **Complete Blood Count (CBC):** To check for infection or polycythemia.
- **Blood Cultures:** If sepsis is suspected.
- **Echocardiography:** If a congenital heart disease is suspected.

6. **Management:**

- **Supportive Care:** Oxygen supplementation, maintenance of normal body temperature, fluid and electrolyte balance.
- **Specific Treatments:** CPAP or mechanical ventilation for severe cases, surfactant therapy for RDS, antibiotics for suspected infection.
- **Monitoring:** Continuous monitoring of respiratory and cardiovascular status.

Condition	Clinical Features	Diagnostic Findings	Initial Management
Respiratory Distress Syndrome (RDS)	Grunting, retractions, nasal flaring More common in preterm infants	Ground-glass appearance on chest X-ray Hypoxemia and hypercapnia on blood gas	Surfactant replacement therapy Mechanical ventilation or CPAP
Transient Tachypnea of the Newborn (TTN)	Tachypnea (>60 breaths/min) Mild retractions, grunting Usually in term or late preterm infants	Hyperinflation and fluid in interlobar fissures on chest X-ray Normal blood gas or mild hypoxemia	Supplemental oxygen Observation and supportive care
Meconium Aspiration Syndrome	Respiratory distress, cyanosis History of meconium-stained amniotic fluid	Patchy infiltrates, atelectasis on chest X-ray Hypoxemia on blood gas	Suctioning of airways Mechanical ventilation if severe Possible ECMO in refractory cases

Neonatal Pneumonia	Tachypnea, retractions, grunting Possible fever or hypothermia Maternal history of infection	Consolidation on chest X-ray Positive blood or CSF cultures	Broad-spectrum antibiotics Supportive respiratory care
Pneumothorax	Sudden onset of distress Decreased breath sounds on affected side Asymmetrical chest movement	Visible pleural line with absence of lung markings on chest X-ray	Needle aspiration or chest tube placement Oxygen supplementation
Congenital Diaphragmatic Hernia (CDH)	Severe respiratory distress Scaphoid abdomen Displaced heart sounds	Bowel loops in chest on chest X-ray Confirmation with ultrasound or MRI	Intubation and gastric decompression Surgical repair after stabilization
Airway Anomalies	Stridor, respiratory distress Difficulty with feeding Symptoms vary with anomaly	Diagnosis often with endoscopy Imaging to define anatomy	Secure airway Surgical correction as indicated
Pulmonary Hypoplasia	Respiratory distress Small chest cavity on examination	Small lung fields on chest X-ray Associated with oligohydramnios	Supportive care with ventilation Manage underlying cause
Pulmonary Hypertension (PPHN)	Severe hypoxemia, cyanosis Poor response to oxygen therapy	Echocardiography to assess pulmonary pressure Hypoxemia on blood gas	Nitric oxide ECMO in refractory cases
Congenital Heart Disease	Cyanosis, heart murmur May have signs of heart failure	Echocardiography for definitive diagnosis Chest X-ray may show cardiomegaly	Prostaglandin E1 to maintain ductal patency Cardiac support, surgical intervention
Metabolic Disorders	Tachypnea, poor feeding May have altered tone or seizures	Blood glucose, electrolytes, and blood gas for metabolic status	Correct metabolic abnormalities Supportive care

Neurological Conditions	Altered level of consciousness Seizures or abnormal movements	Neuroimaging for structural anomalies EEG if seizures suspected	Secure airway Neuroprotective measures
Hematologic Issues (Polycythemia /Anemia)	Ruddy complexion or pallor Tachypnea, lethargy	CBC for hematocrit or hemoglobin levels	Partial exchange transfusion for polycythemia Transfusion for anemia
Birth Asphyxia	Poor respiratory effort Low Apgar scores Need for resuscitation	Blood gas for acid-base status Clinical history and examination	Resuscitation Supportive care

7. a) Management of neonatal seizures.

Clinical History and Examination:

- Detailed prenatal, perinatal, and postnatal history.
- Neurological examination to identify the seizure type and possible etiology.

Investigations required in neonates with seizures:

• Essential investigations (required in all with few exceptions):

- Blood sugar level assessment.
- Serum sodium and calcium levels to check for electrolyte imbalances.
- Cerebrospinal fluid (CSF) examination to rule out infections or hemorrhages.
- Cranial ultrasound to assess brain structure and potential hemorrhages or cysts.
- Electroencephalography (EEG) or amplitude-integrated EEG to monitor electrical activity in the brain.
- Toxicology screening if drug exposure is suspected

- **Additional investigations:**

- Hematocrit, especially if the neonate is plethoric or there's a risk of polycythemia.
- Serum bilirubin if jaundice is present, indicating possible hyperbilirubinemia.
- Serum magnesium levels, as hypomagnesemia can contribute to seizures.
- Arterial blood gas and anion gap to evaluate for metabolic acidosis or other disturbances.
- Imaging with CT or MRI if no cause is found after initial investigations to look for structural abnormalities.
- TORCH screen for congenital infections which can be a cause of seizures.
- Work-up for inborn errors of metabolism which can present with seizures.

- **Neuroimaging:**

- Cranial ultrasound as a first-line imaging modality, especially useful for intracranial hemorrhages and hydrocephalus in preterm infants.
- Magnetic Resonance Imaging (MRI) to identify cerebral anomalies, white matter injury, or signs of hypoxic-ischemic injury.
- Computed Tomography (CT) scan if MRI is not available or in emergency situations to quickly assess for hemorrhage or major structural abnormalities.

- **Electroencephalogram (EEG):**

- Conventional EEG to characterize seizure activity, identify the focus, and assess for background brain activity.
- Amplitude-integrated EEG (aEEG) for continuous monitoring in the neonatal intensive care unit (NICU).

- **Genetic and Metabolic Testing:**

- If a metabolic or genetic disorder is suspected based on clinical and laboratory findings, specific tests for metabolic disorders and genetic analysis may be indicated.

Management protocol for neonate with seizures:

• ***Initial steps:***

- Identify and characterize the seizure.
- Optimize breathing, circulation, and temperature.
- Administer oxygen if needed.
- Secure IV access and take blood samples for baseline investigations.
- Administer 2 mL/kg of 10% dextrose as a bolus if blood sugar is low.
- Follow with a continuous infusion of glucose.
- If blood sugar is normal, administer 2 mL/kg of calcium gluconate (10%) under cardiac monitoring.

• ***Pharmacological treatment if seizures persist:***

- Start with phenobarbitone at a dose of 20 mg/kg IV over 20 minutes.
- If seizures continue, repeat phenobarbitone in 10 mg/kg doses up to 40 mg/kg.
- If seizures still persist, administer phenytoin at 20 mg/kg IV slowly over 20 minutes under cardiac monitoring.
- Repeat phenytoin in 10 mg/kg doses if needed.
- If seizures are not controlled, consider lorazepam or midazolam bolus and infusion.
- Once seizures are controlled, wean antiepileptic drugs (AEDs) slowly and maintain with phenobarbitone.

• ***Treatment of Underlying Cause:***

- Antibiotics for suspected bacterial infections.
- Correction of metabolic imbalances (e.g., glucose, calcium, magnesium).

- Management of hypoxic-ischemic encephalopathy (HIE) with therapeutic hypothermia if within the therapeutic window.

- **Long-term Management:**

- Regular follow-up with neurology for ongoing seizure management and adjustment of AEDs.
- Developmental surveillance and early intervention services to address potential neurodevelopmental issues.
- Family support and genetic Counselling if a hereditary condition is identified.

- **Monitoring and Adjustments:**

- Continuous or repeated EEG monitoring to assess treatment efficacy and guide AED adjustments.
- Monitoring for side effects of AEDs, including hepatic, hematologic, and nutritional monitoring.



7. b) Surgical emergencies in newborn

<i>Surgical Emergency</i>	<i>Key Features</i>	<i>Presentation</i>	<i>Management</i>
<i>Congenital Diaphragmatic Hernia (CDH)</i>	Herniation of abdominal contents into thoracic cavity	Respiratory distress, scaphoid abdomen	Surgical repair after stabilization
<i>Omphalocele and Gastroschisis</i>	Omphalocele: Herniation with membrane; Gastroschisis: Herniation without membrane	Abdominal contents visible at birth	Surgical correction, often staged
<i>Intestinal Atresia</i>	Congenital absence or closure of intestine	Abdominal distension, vomiting, failure to pass meconium	Surgical resection and anastomosis
<i>Necrotizing Enterocolitis (NEC)</i>	Inflammatory condition of intestines, mainly in preterms	Feeding intolerance, abdominal distension, bloody stools	Surgery for severe cases with perforation/necrosis
<i>Hirschsprung Disease</i>	Absence of ganglion cells in distal colon	Failure to pass meconium, abdominal distension, vomiting	Surgical resection of aganglionic segment
<i>Meconium Ileus</i>	Obstruction with thick meconium, associated with cystic fibrosis	Abdominal distension, failure to pass meconium	Surgery if unresponsive to conservative management
<i>Tracheoesophageal Fistula and Esophageal Atresia</i>	Abnormal connection between trachea and esophagus	Frothy saliva, coughing, choking, respiratory distress	Surgical correction
<i>Imperforate Anus</i>	Absence of normal anal opening	Failure to pass meconium, distended abdomen	Surgical creation of anal opening, possibly staged
<i>Malrotation with Volvulus</i>	Abnormal intestinal rotation leading to obstruction	Bilious vomiting, abdominal pain, distension	Emergency surgery to untwist/resect bowel
<i>Ovarian and Testicular Torsion</i>	Twisting of ovaries or testes	Abdominal or scrotal pain, swelling	Urgent surgical intervention

- **Early Diagnosis:** Early recognition and diagnosis are critical for the successful management of these conditions.

- **Interdisciplinary Approach:** Management often requires a team approach involving neonatologists, pediatric surgeons, and other specialists.
- **Postoperative Care:** Post-surgical care and monitoring are essential for recovery and long-term outcomes.

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8. a) Principles of management of autistic spectrum disorders

- ❖ Each child with ASD is unique, and what works for one child may not work for another.
 - ❖ The management plan should be highly individualized, taking into account the child's strengths, weaknesses, and specific challenges.
1. **Early Intervention:** Early diagnosis and intervention are crucial. Interventions are more effective when started at a younger age.
 2. **Behavioral Therapies:**
 - Applied Behavior Analysis (ABA): A widely used therapy that improves social, communication, and learning skills through positive reinforcement.
 - Early Start Denver Model (ESDM): A behavioral therapy for children with ASD aged 12-48 months, which integrates ABA techniques with developmental and relationship-based approaches.
 3. **Educational Therapies:**
 - Structured teaching environments can help children with ASD learn life skills and academic subjects.
 - Individualized Education Programs (IEPs) are tailored to each child's unique needs.
 4. **Speech Therapy:** Addresses challenges with language and communication, helping children improve their ability to express themselves verbally and nonverbally.
 5. **Occupational Therapy:** Focuses on improving daily living skills, such as dressing, eating, and interacting with others.
 6. **Social Skills Training:** Helps children learn how to interact more effectively with others and understand social cues.

7. **Parent Training and Support:** Educating and involving parents in the therapeutic process is vital. This includes training on how to interact and communicate with their child effectively.
8. **Pharmacological Treatment:** While there is no medication that can cure ASD, certain medications can help manage symptoms like anxiety, depression, or hyperactivity.
9. **Dietary and Nutritional Approaches:** Some parents opt for dietary strategies, such as gluten-free or casein-free diets, although their effectiveness is not universally supported by scientific evidence.
10. **Complementary and Alternative Therapies:** Some families explore therapies like music therapy, animal therapy, or acupuncture, though these should be approached with caution and in conjunction with more traditional therapies.
11. **Technology-Aided Instruction and Intervention:** Utilizing technology, including apps and software, can support learning and skill development.
12. **Regular Monitoring and Adaptation of Strategies:** Continuous assessment of the child's progress and adjusting interventions as needed.
13. **Collaboration Among Care Providers:** Effective management often involves a team of professionals, including pediatricians, neurologists, psychologists, and educators, working together.
14. **Inclusion and Mainstreaming:** Whenever possible, including children with ASD in mainstream classrooms and activities, with appropriate support, to promote socialization and integration.
15. **Transition Planning for Adolescence and Adulthood:** Preparing for transitions, including the shift to adulthood, is an important aspect of long-term care.

8. b) Vaccine hesitancy

- ❖ Vaccine hesitancy refers to the delay in acceptance or refusal of vaccines despite the availability of vaccination services
- ❖ It is a complex and context-specific phenomenon, varying across time, place, and vaccines
- ❖ Understanding and addressing vaccine hesitancy is crucial for maintaining high vaccination coverage and ensuring public health
- ❖ Vaccine hesitancy is a significant barrier to achieving widespread vaccine coverage and requires a multifaceted approach to address

- ❖ It involves understanding the root causes of hesitancy, engaging with communities, providing accurate information, and building trust in vaccines and the healthcare system
- ❖ The role of healthcare providers, public health authorities, and community leaders is crucial in this effort.

1. ***Causes of Vaccine Hesitancy:***

- ***Misinformation and Lack of Knowledge:*** Spread of false information about vaccines, leading to misconceptions.
- ***Cultural, Religious, and Philosophical Beliefs:*** Beliefs that discourage vaccination.
- ***Safety Concerns:*** Fears about potential side effects or adverse events.
- ***Distrust in Healthcare System or Government:*** Skepticism about vaccine efficacy or motives behind vaccination campaigns.
- ***Complacency:*** Perception that the risk of vaccine-preventable diseases is low.

2. ***Impact of Vaccine Hesitancy:***

- ***Outbreaks of Vaccine-Preventable Diseases:*** Reduced herd immunity, leading to outbreaks.
- ***Increased Healthcare Costs:*** Treating preventable diseases is often more expensive than vaccination.
- ***Public Health Risks:*** Endangers vulnerable populations, including those who cannot be vaccinated.

3. ***Strategies to Address Vaccine Hesitancy:***

- ***Education and Awareness Campaigns:*** Providing accurate, evidence-based information about vaccines.
- ***Engaging with Communities:*** Understanding specific concerns and cultural contexts.
- ***Building Trust:*** Transparent communication from healthcare providers and authorities.
- ***Involvement of Influencers:*** Utilizing community leaders or celebrities to promote vaccination.
- ***Policy Interventions:*** Implementing policies that encourage vaccination, such as school entry requirements.

4. **Role of Healthcare Providers:**

- **Effective Communication:** Addressing concerns and questions about vaccines.
- **Recommendation:** A strong recommendation from a healthcare provider is a key influencer in the decision to vaccinate.
- **Patient-Centered Approach:** Understanding individual patient's concerns and providing tailored information.

5. **Monitoring and Research:**

- **Surveillance of Vaccine Attitudes:** Tracking public sentiment towards vaccines.
- **Research on Vaccine Safety and Efficacy:** Ongoing research to ensure and communicate vaccine safety.

6. **Global and Local Initiatives:**

- **World Health Organization (WHO) Initiatives:** Global strategies to enhance vaccine confidence.
- **Local Health Campaigns:** Targeted efforts based on the specific needs of communities.

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9. a) **Clinical presentation & management of Henoch-Schonlein purpura.**

Henoch-Schönlein Purpura (HSP), also known as IgA vasculitis, is a small vessel vasculitis that primarily affects children.

It typically presents with palpable purpura, joint pain, abdominal pain, and kidney involvement.

Clinical Features

- **Dermatological:** Palpable purpura, predominantly in lower extremities.
- **Gastrointestinal:** Abdominal pain, often acute, diffuse, and colicky.
- **Musculoskeletal:** Arthritis or arthralgia affecting large joints like knees and ankles.
- **Renal:** Hematuria, proteinuria, and nephritic syndrome in severe cases.

- **Other:** Rarely, involvement of lungs, CNS, and scrotum.

Diagnosis

- **Classification Criteria:** Palpable purpura in the absence of coagulopathy or thrombocytopenia, along with one or more of the following: abdominal pain, arthritis or arthralgia, and IgA deposition in biopsy.
- **Laboratory Tests:** Elevated ESR, CRP, and sometimes thrombocytosis.
- **Biopsy:** Predominant IgA deposition in affected tissue.
- **Imaging:** Ultrasound for abdominal complications; renal biopsy in severe renal involvement.

Management:

The management of HSP is primarily supportive, focusing on symptom relief and monitoring for complications.

Corticosteroids are used for severe manifestations, especially with significant renal or gastrointestinal involvement.

Regular follow-up is essential to monitor for renal involvement and other complications.

1. Supportive Care:

- **Rest and Observation:** Recommended during the acute phase, especially if joint pain is significant.
- **Pain Management:** NSAIDs (e.g., ibuprofen) for joint and abdominal pain, but with caution in renal involvement.
- **Hydration and Nutrition:** Adequate hydration and a balanced diet.

2. Corticosteroids:

- Indicated for severe or persistent abdominal pain, significant joint pain, or renal involvement.
- Prednisone is commonly used.
- The duration of therapy varies based on symptoms and response to treatment.

3. Management of Renal Involvement:

- **Mild Proteinuria and Hematuria:** Usually require only observation and regular monitoring.

- **Significant Renal Involvement:** May require corticosteroids, and in severe cases, immunosuppressive agents like cyclophosphamide or azathioprine.
 - **Regular Monitoring:** Renal function and urine analysis should be monitored regularly to detect early signs of progression.
4. **Gastrointestinal Involvement:**
- Corticosteroids for severe abdominal pain or complications like intussusception.
 - Monitoring for signs of bowel perforation or intussusception.
5. **Skin Care:**
- Good skin hygiene to prevent secondary infections.
 - Topical treatments are not usually necessary for the purpura.
6. **Patient and Family Education:**
- Educate about the nature of the disease, its course, and symptoms to monitor.
 - Reassurance about the generally good prognosis.
7. **Follow-Up:**
- Regular follow-up to monitor for recurrence or complications.
 - Blood pressure and renal function monitoring are important.

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9. b) Risk stratification in Acute Lymphoblastic Leukemia (ALL) in children

- ❖ Risk stratification in Acute Lymphoblastic Leukemia (ALL) in children is a critical process that guides treatment intensity.
- ❖ It involves categorizing patients based on various prognostic factors to predict the risk of relapse and to tailor therapy accordingly.
- ❖ Risk stratification in pediatric ALL is essential for optimizing treatment, balancing the need for effective therapy against the risk of long-term treatment-related side effects.

Here's an overview of the factors involved in risk stratification:

1. **Age at Diagnosis:**

- Children aged 1-9 years have a more favorable prognosis.
- Infants (<1 year) and older children (≥ 10 years) are at higher risk.

2. **White Blood Cell (WBC) Count at Diagnosis:**

- High WBC count at diagnosis ($>50,000/\mu\text{L}$) is associated with a higher risk.

3. **Immunophenotype:**

- B-cell lineage ALL generally has a better prognosis than T-cell lineage.

4. **Genetic Abnormalities:**

- Certain chromosomal abnormalities confer different risks:

- **Favourable:** Hyper diploidy (>50 chromosomes) and $t(12;21)(p13;q22)$ (ETV6-RUNX1).
- **Unfavourable:** $t(9;22)(q34;q11.2)$ (BCR-ABL1), $t(4;11)(q21;q23)$ (MLL rearrangements), hypodiploidy (fewer than 44 chromosomes).

5. **Response to Initial Therapy:**

- Early response to therapy, measured by minimal residual disease (MRD):
 - Good Prognosis: Negative MRD (no detectable leukemia cells) after induction therapy.
 - Poor Prognosis: Positive MRD indicates resistant disease.

6. **Central Nervous System (CNS) Involvement:**

- CNS involvement at diagnosis is a high-risk feature.

7. **Testicular Involvement in Males:**

- Testicular involvement at diagnosis can indicate a higher risk.

8. **Race and Ethnicity:**

- Some studies suggest variations in risk and outcomes among different racial and ethnic groups.

Risk Groups:

Based on these factors, patients with ALL are typically classified into three risk groups:

1. **Standard Risk (SR):**

- Younger age (1-9 years), lower WBC count, favorable genetics, and good response to initial therapy.

2. **Intermediate Risk (IR):**

- Patients who do not fit clearly into SR or HR categories.

3. **High Risk (HR):**

- Older age (≥ 10 years), high WBC count, unfavorable genetics, poor response to initial therapy, CNS/testicular involvement.

Treatment Implications:

- **Standard Risk:** Less intensive chemotherapy.
- **High Risk:** More intensive chemotherapy, possible stem cell transplantation, and targeted therapies.

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10. a) Autoimmune encephalitis

- Autoimmune encephalitis is a complex, multi-faceted neurological disorder.
- It involves inflammation of the brain tissue triggered by an aberrant immune response.
- Early diagnosis is crucial for effective management and prognosis.

Clinical Presentation

- Altered mental status
- Seizures
- Movement disorders
- Psychiatric symptoms
- Memory deficits
- Speech disturbances

Laboratory Investigations

- **Complete Blood Count (CBC):** To rule out infection.
- **C-Reactive Protein (CRP) and Erythrocyte Sedimentation Rate (ESR):** Inflammatory markers.
- **Lumbar Puncture:** For CSF analysis.
 - Oligoclonal bands
 - Elevated protein
 - Pleocytosis

Imaging Studies

- **MRI Brain:** To assess structural abnormalities.
 - T2/FLAIR hyperintensities
 - Limbic system involvement
- **CT Scan:** Less sensitive but used in emergency settings.

Autoantibody Testing

- **NMDA Receptor Antibodies**
- **LGI1, CASPR2 Antibodies**
- **GABA Receptor Antibodies**
- **AMPA Receptor Antibodies**

Electroencephalogram (EEG)

- To evaluate for seizure activity or other abnormal electrical discharges.
- May show non-specific slowing or epileptiform discharges.

Additional Tests

- **PET/CT:** To identify metabolic changes in the brain.
- **Autoimmune Panel:** To rule out systemic autoimmune diseases.

Differential Diagnosis

- Viral encephalitis
- Neurodegenerative disorders
- Psychiatric illnesses

- Metabolic encephalopathy

Confirmatory Tests

- Positive autoantibody test along with clinical and imaging findings.
- Improvement with immunotherapy can also be confirmatory.

Management

1. **Corticosteroids:**

- **Methylprednisolone:**

- Standard dosage is 20-30 mg/kg/day intravenously (IV), with a maximum of 1 gram per day.
- Administered for 3-5 days.
- Dosage may be adjusted based on response and side effects.

- **Prednisone/Prednisolone** (oral):

- Used for tapering after IV methylprednisolone.
- Dosage: 1-2 mg/kg/day, with a gradual taper over weeks to months.

2. **Intravenous Immunoglobulin (IVIG):**

- Standard dosage is 0.4 g/kg/day IV for 5 days.
- Total dose of 2 g/kg, divided over the course of treatment.
- Close monitoring for allergic reactions and side effects is necessary.

3. **Plasma Exchange (PLEX):**

- Typically, 5-7 exchanges are performed over 10-14 days.
- Each exchange involves removing 1-1.5 plasma volumes.
- Used in severe cases or when there is no response to steroids and IVIG.

Additional Considerations:

- **Monitoring:** Patients receiving these therapies require close monitoring for potential side effects. This includes monitoring blood pressure, blood sugar levels (especially with corticosteroids), and signs of infection.

- **Adjustments for Age and Weight:** Dosages should be carefully calculated based on the patient's age and weight, particularly in very young children.
- **Response to Therapy:** The response to therapy should be regularly assessed. Lack of improvement may necessitate a switch to second-line therapies.
- **Tapering of Steroids:** When tapering steroids, it's important to do so gradually to avoid adrenal insufficiency.
- **Infection Prophylaxis:** Patients receiving immunosuppressive therapy may require prophylaxis against specific infections, such as Pneumocystis jirovecii pneumonia (PCP).

Second-Line Therapies:

- Initiated if there is inadequate response to first-line therapies.
- **Rituximab:** A monoclonal antibody that depletes B-cells.
- **Cyclophosphamide:** An immunosuppressive agent used in severe cases.

Seizure Management:

- Antiepileptic drugs (AEDs) are used to control seizures.
- Continuous EEG monitoring may be necessary in severe cases.

Treatment of Underlying Cause:

- If a tumor (e.g., teratoma) is associated with AE, surgical removal is often necessary.
- Addressing any other identified triggers or underlying conditions.

Supportive Care:

- Rehabilitation services including physical therapy, occupational therapy, and speech therapy.
- Psychological support and psychiatric care for behavioral or mood disturbances.
- Educational support to address cognitive deficits.

Long-Term Immunosuppression:

- For patients with recurrent or refractory disease.
- Options include azathioprine, mycophenolate mofetil, or methotrexate.

Monitoring and Follow-Up:

- Regular follow-up to monitor for disease recurrence and side effects of treatment.
- Repeat imaging and laboratory tests as indicated.

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10. b) Immunization in adolescents

Key vaccines recommended for adolescents:

1. Tetanus, Diphtheria, and Pertussis (Tdap):

- **Age:** Recommended at 11-12 years.
- **Dose:** Single dose.
- **Purpose:** Booster to protect against tetanus, diphtheria, and pertussis.

2. Human Papillomavirus (HPV) Vaccine:

- **Age:** Recommended starting at 11-12 years; can be given starting at age 9.
- **Dose:**
 - Two doses for those who start the series before their 15th birthday, given 6-12 months apart.
 - Three doses for those who start on or after their 15th birthday, with the second dose 1-2 months after the first, and the third dose 6 months after the first.
- **Purpose:** Protects against HPV-related cancers and genital warts.

3. Meningococcal Conjugate Vaccine (MenACWY):

- **Age:** First dose at 11-12 years, with a booster at 16 years.
- **Dose:** Two doses.
- **Purpose:** Protects against meningococcal disease caused by serogroups A, C, W, and Y.

4. Meningococcal B Vaccine (MenB):

- **Age:** May be given to teens aged 16-18 years, based on individual decision-making.
- **Dose:**
 - Two or three doses depending on the brand (Bexsero: 2 doses, Trumenba: 3 doses).
- **Purpose:** Protects against meningococcal disease caused by serogroup B.

5. **Influenza Vaccine:**

- **Age:** Annually for all adolescents.
- **Dose:** One dose each year.
- **Purpose:** Protects against seasonal influenza.

6. **Measles, Mumps, and Rubella (MMR):**

- **Age:** If not given in childhood, should be administered in adolescence.
- **Dose:** Two doses.
- **Purpose:** Protects against measles, mumps, and rubella.

7. **Varicella Vaccine:**

- **Age:** If not given in childhood, recommended for adolescents without evidence of immunity.
- **Dose:** Two doses.
- **Purpose:** Protects against chickenpox.

8. **Hepatitis A Vaccine:**

- **Age:** For adolescents not vaccinated as children.
- **Dose:** Two doses, 6 months apart.
- **Purpose:** Protects against hepatitis A.

9. **Hepatitis B Vaccine:**

- **Age:** For adolescents not vaccinated as children.
- **Dose:** Three doses over 6 months.
- **Purpose:** Protects against hepatitis B.

Additional Considerations:

- **Catch-Up Vaccinations:** Adolescents who missed earlier vaccines should receive catch-up vaccinations.
- **Consent and Education:** It's important to educate adolescents and their guardians about the benefits and potential side effects of vaccines.
- **Special Circumstances:** Additional vaccines may be recommended for adolescents with certain health conditions like pregnancy, lifestyles, or travel plans.

Vaccination for consideration in adolescent pregnancies

1. **Influenza Vaccine:**

- **Timing:** Any trimester.
- **Type:** Inactivated influenza vaccine (IIV) or recombinant influenza vaccine (RIV).
- **Purpose:** Protects against seasonal influenza, which can be more severe in pregnant women.

2. **Tetanus, Diphtheria, and Pertussis (Tdap) Vaccine or TT :**

- **Timing:** Ideally between 27 and 36 weeks of gestation, in every pregnancy.
- **Type:** Tdap or TT in public sector.
- **Purpose:** Protects against tetanus, diphtheria, and pertussis. Pertussis vaccination in pregnancy helps protect the newborn through passive transfer of antibodies.

3. **COVID-19 Vaccine:**

- **Timing:** Any trimester, including during preconception period.
- **Type:** mRNA vaccines (e.g., Pfizer-BioNTech, Moderna) are commonly recommended.
- **Purpose:** Protects against COVID-19, which can have more severe effects in pregnant women.

Vaccines to Avoid During Pregnancy:

- **Live Vaccines:** Such as MMR (measles, mumps, rubella), varicella (chickenpox), and live attenuated influenza vaccine (LAIV).

- **Other Live Vaccines:** Such as the nasal spray flu vaccine and the yellow fever vaccine, should be avoided unless the benefits outweigh the risks (e.g., in the case of travel to areas with high yellow fever risk).

Additional Considerations:

- **Hepatitis B Vaccine:** If a pregnant woman is at high risk for hepatitis B infection (e.g., healthcare worker, sexually active with multiple partners), vaccination is recommended.
- **Pneumococcal Vaccine:** Recommended for pregnant women with certain chronic conditions that increase the risk of pneumococcal disease.
- **Pre-Pregnancy Vaccination:** Women planning a pregnancy should ensure they are up to date with recommended vaccines, particularly MMR and varicella, to avoid infections that can be harmful during pregnancy.

Counselling and Consent:

- **Risk-Benefit Analysis:** Healthcare providers should discuss the benefits and potential risks of vaccines with pregnant women.
- **Informed Consent:** Pregnant women should provide informed consent after understanding the importance of vaccination for their health and their baby's health.

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Paper 3

1. a) Approach to child with upper airway obstruction

The approach to a child with upper airway obstruction requires prompt assessment and intervention, as it can be a life-threatening emergency.

The management involves identifying the cause of the obstruction, assessing the severity, and providing immediate treatment.

1. *Initial Assessment:*

- **Airway:** Check for patency of the airway.
- **Breathing:** Assess breathing quality, rate, and effort.
- **Circulation:** Evaluate heart rate, pulse, and skin color.
- **Consciousness:** Check the level of consciousness.
- **Signs of Severe Obstruction:** Stridor, retractions, cyanosis, altered mental status, inability to speak or cry.

2. *History and Physical Examination:*

- **History:** Ask about onset, duration, associated symptoms (cough, fever, voice change), choking events, and medical history.
- **Physical Exam:** Look for foreign bodies, swelling, trauma, or signs of infection.

3. *Severity Assessment:*

- **Mild Obstruction:** Child is able to speak or cry, may have stridor only when agitated.
- **Moderate Obstruction:** Obvious stridor at rest, increased work of breathing, retractions.
- **Severe Obstruction:** Marked stridor, cyanosis, altered mental status, fatigue.

4. *Management Based on Severity:*

- **Mild to Moderate Obstruction:**
 - Position for comfort, usually sitting upright.
 - Administer humidified oxygen if available.
 - Consider nebulized epinephrine if stridor is present.

- Corticosteroids (e.g., dexamethasone) can be beneficial in cases like croup.
- **Severe Obstruction:**
 - Immediate airway intervention is critical.
 - Call for emergency support (e.g., advanced airway management team).
 - Prepare for potential intubation or surgical airway if necessary.
 - Do not attempt blind finger sweeps in the case of suspected foreign body aspiration.

5. **Specific Interventions:**

- **Foreign Body Aspiration:** Heimlich maneuver (if age-appropriate) or back blows and chest thrusts in infants.
- **Anaphylaxis:** Administer epinephrine immediately.
- **Infection (e.g., Epiglottitis):** Secure airway, start antibiotics, and provide supportive care.

6. **Monitoring and Supportive Care:**

- Continuous monitoring of vital signs and oxygen saturation.
- IV access for fluid and medication administration.
- Admit to hospital if necessary, especially for observation after a severe episode or if the cause is unclear.

7. **Prevention and Education:**

- Educate caregivers about avoiding small objects that can be aspirated.
- Discuss the importance of timely vaccination (e.g., Haemophilus influenzae type b vaccine to prevent epiglottitis).

Differential diagnoses in a child with upper airway obstruction:

Conditions	Key Features	Management
Infectious Causes		
<i>Acute Epiglottitis</i>	Sudden onset, drooling, dysphagia, high fever, toxic appearance	Emergency airway management, IV antibiotics,

		corticosteroids, possible intubation
<i>Bacterial Tracheitis</i>	High fever, barking cough, stridor, often follows a viral URI	Hospitalization, IV antibiotics, possible intubation, supportive care
<i>Retropharyngeal Abscess</i>	Fever, neck stiffness, dysphagia, muffled voice, bulging posterior pharyngeal wall	IV antibiotics, surgical drainage if necessary, airway monitoring
<i>Tonsillitis/Adenoiditis</i>	Sore throat, difficulty swallowing, muffled voice, enlarged tonsils	Oral antibiotics, pain management, tonsillectomy in recurrent cases
Non-Infectious Causes		
<i>Allergic Reactions/Anaphylaxis</i>	Sudden onset, hives, swelling (lips, tongue), wheezing, allergen exposure	Epinephrine, antihistamines, corticosteroids, airway monitoring, avoid allergen
<i>Foreign Body Aspiration</i>	Sudden onset of coughing, choking, gagging, unilateral wheezing or decreased breath sounds	Bronchoscopy for removal, emergency intervention if severe
<i>Laryngomalacia</i>	Stridor worsening with crying, feeding, supine position, common in infants	Observation in mild cases, surgical intervention in severe cases (supraglottoplasty)
<i>Vocal Cord Dysfunction</i>	Episodic stridor, voice changes, stress or exercise triggered	Speech therapy, breathing techniques, stress management
<i>Subglottic Stenosis</i>	History of prolonged intubation, biphasic stridor, chronic symptoms	Endoscopic evaluation, possible surgical intervention (e.g., laryngotracheal reconstruction)
Traumatic Causes		
<i>Thermal or Chemical Burn</i>	Ingestion or exposure to caustic substance, pain, voice change, drooling	Airway stabilization, endoscopic assessment, IV fluids, pain management, referral to specialist
<i>External Trauma to Neck</i>	History of trauma, pain, swelling, bruising in neck area	Airway assessment, imaging, surgical consultation if necessary
Neoplastic Causes		

<i>Laryngeal Papillomatosis</i>	Progressive/ recurrent hoarseness, stridor, respiratory distress	Surgical removal of papillomas, adjuvant therapy (e.g., cidofovir), regular followup
<i>Tumors (e.g., lymphoma)</i>	Progressive symptoms, possible mass or swelling, systemic symptoms (weight loss, night sweats)	Biopsy for diagnosis, oncology referral, chemotherapy/radiation as indicated
Immunological Causes		
<i>Croup (Viral Laryngotracheobronchitis)</i>	Barking cough, stridor, hoarseness, viral URI symptoms	Nebulized epinephrine for severe cases, corticosteroids, supportive care
<i>Angioedema</i>	Rapid onset of swelling (face, lips, tongue), history of similar episodes	Epinephrine, antihistamines, corticosteroids, airway monitoring, identify and avoid triggers

- Management strategies are tailored to the severity of the condition and the individual patient.
- In all cases of severe upper airway obstruction, securing the airway is the primary concern.
- Some conditions, like anaphylaxis and acute epiglottitis, require immediate emergency intervention.

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1. b) Types of supraventricular tachycardia and management of SVT with circulatory failure

Classification of Arrhythmias

- **Stable Arrhythmias**
 - Sinus Tachycardia
 - Atrial Fibrillation
 - Atrial Flutter
 - AVNRT (Atrioventricular Nodal Reentrant Tachycardia)

- **Unstable Arrhythmias**

- Ventricular Tachycardia
- Ventricular Fibrillation
- Supraventricular Tachycardia with hemodynamic instability

Types of Supraventricular Tachycardia (SVT) and ECG Changes

- **Atrioventricular Reciprocating Tachycardia (AVRT)**
 - ECG: Delta wave, wide QRS complex
- **Atrioventricular Nodal Reentrant Tachycardia (AVNRT)**
 - ECG: Narrow complex tachycardia, P waves hidden in T waves
- **Atrial Tachycardia**
 - ECG: Abnormal P wave axis, similar ventricular rates

Management of Unstable Arrhythmias

- **Initial Steps**
 - Immediate ECG
 - Hemodynamic assessment
 - IV access
- **Pharmacological Interventions**
 - Amiodarone or Procainamide for VT
 - Adenosine for unstable SVT
- **Electrical Cardioversion**
 - Synchronized cardioversion for unstable VT or SVT
- **Advanced Measures**
 - Emergency ablation for refractory cases
 - Temporary pacing for complete heart block

SVT with circulatory failure

The primary goal is to restore normal heart rhythm and ensure adequate circulation.

1. **Initial Assessment and Stabilization:**

- Assess ABCs (Airway, Breathing, Circulation).
 - Establish IV/IO (intraosseous) access.
 - Continuous cardiac monitoring and pulse oximetry.
 - Obtain vital signs, including blood pressure.
2. **Vagal Maneuvers** (if the child is stable enough to attempt):
- Ice to face (diving reflex) in infants.
 - Valsalva maneuver in older children.
3. **Adenosine** (if vagal maneuvers are ineffective or not feasible):
- **First Dose:** 0.1 mg/kg rapid IV push (maximum 6 mg).
 - **Second Dose** (if needed): 0.2 mg/kg rapid IV push (maximum 12 mg).
 - Administer as close to the heart as possible, followed by a rapid saline flush.
 - Brief period of asystole is common after administration.
4. **Synchronized Cardioversion** (if adenosine is ineffective or if the child is hemodynamically unstable):
- **Initial Dose:** 0.5-1 J/kg.
 - **Second Dose** (if first is ineffective): 2 J/kg.
 - Sedation/analgesia as per protocol if the child is conscious.
5. **Advanced Pharmacotherapy** (if cardioversion is unsuccessful or not feasible):
- **Amiodarone:**
 - Load: 5 mg/kg over 20-60 minutes IV.
 - Maintenance: 5-15 µg/kg/minute.
 - **Procainamide:**
 - Load: 15 mg/kg over 30-60 minutes IV (not to exceed 50 mg/min).
 - Maintenance: 20-80 µg/kg/minute.
 - **Esmolol** (if beta-blocker is considered):
 - Load: 500 µg/kg over 1 minute.

- Maintenance: 50-200 µg/kg/minute.

6. **Supportive Care:**

- Oxygen if hypoxic.
- Treat underlying causes (e.g., fever, pain, dehydration).
- Monitor for complications like heart failure or thromboembolism.

7. **Post-Conversion Management:**

- Continuous monitoring for recurrence.
- Electrolyte and acid-base balance correction.
- Consider transfer to higher level of care if needed.

8. **Long-Term Management:**

- Cardiology referral for further evaluation and management.
- Consideration of long-term antiarrhythmic therapy.
- Rapid identification and treatment of circulatory failure are crucial.
- The choice between adenosine and immediate cardioversion depends on the patient's hemodynamic stability.
- Close monitoring for adverse effects, especially with antiarrhythmic drugs, is necessary.
- In all cases, the management should be tailored to the individual patient, considering underlying conditions and the response to initial treatments.

Post-event monitoring and long-term management are essential for preventing recurrence and addressing any underlying conditions.

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2.a) Evaluation of medical morbidities in Down syndrome.

Morbidity	Evaluation	Management
Congenital Heart Defects	Echocardiogram, ECG, pediatric cardiologist assessment.	Surgical interventions if necessary, ongoing cardiac monitoring, management of heart failure.